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# TUBERCULOSIS AND THE "SOCIAL COMPLEX" IN GLASGOW

BY

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In recent years, the high mortality and incidence of respiratory tuberculosis in Scotland have caused much concern. This concern became intensified after the war, when Scottish tuberculosis rates remained at their high war-time level or rose even higher; in other countries which had suffered early war-time increases in tuberculosis the declining rates of the 1930s returned. Neither discussion nor investigation has succeeded in identifying the cause of this dissimilarity, and without precise knowledge of the environmental and sociological background of tuberculosis, such discussion must inevitably be speculative.

In 1949 and 1950 the total mortality from respiratory tuberculosis in Scotland fell, but notifications continued to mount, and even in 1950 the new cases, though slightly fewer than in 1949, were more numerous than in any year in the previous three decades. Apparently the considerable fall in mortality did not signify a similar fall in incidence. Moreover, in the younger age groups, the age-specific rates of death as well as of notification reveal disturbing increases; and these increases at the susceptible ages were manifest even before the war (Scotland, Department of Health, 1951). Any attempt to understand these trends against their background and to determine the factors operative in them must include a detailed investigation of tuberculosis in Glasgow. Within this city lives one-fifth of the population of Scotland, contributing one-third of the Scottish tuberculosis cases and deaths; and surrounding the city are burghs and urbanized areas containing almost another fifth of the population, among whom similar tuberculosis rates prevail.

Before 1939 the death and notification rates in Glasgow did not display the same pattern as those of the rest of Scotland. In the 1930s tuberculosis in Glasgow rose and fell irregularly, always remaining at a high level with little evidence of any real decline. By contrast, both mortality and notification rates in Scotland as a whole fell fairly steadily. War-time increases in tuberculosis were greater in Glasgow than in the rest of Scotland, and the end of the war brought no decline. In 1949 and 1950 the Glasgow mortality fell, and this was a major factor in the total Scottish reduction. At the same time the notifications in Glasgow fell, and this influenced the Scottish total in 1950.

The present tuberculosis position in Scotland is not such as to encourage complacency. The recent drop in the death rate does not necessarily imply a fall in prevalence. Improved methods of treatment have undoubtedly prolonged the survival of notified cases, but while the spread of infection continues and so many new cases are notified annually, tuberculosis cannot be considered to be under control. The urgent

need now is precise knowledge of the environmental background of the disease, of the origins of the Scottish trends, and of the causes of the high Glasgow rates. The vague general belief that tuberculosis is a social disease is of little aid in preventing its further spread or in ameliorating the conditions most closely associated with its incidence. General references to "social conditions" do not lead to specific actions. To achieve control, the influence of different social factors upon mortality and incidence must be established, and the effects of past circumstances and present policy assessed.

A number of statistical studies have been undertaken to demonstrate the influence of social conditions on tuberculosis, and some have sought to isolate specific environmental circumstances. Greenwood (1926) investigated the connexion between housing and respiratory tuberculosis in the boroughs of London. Hart and Wright (1939) demonstrated the relationship between this disease and a number of social indices in the cities and counties of England. McKinlay (1947) examined overcrowding and tuberculosis mortality changes in Scotland in pre-war years. Stein (1950) showed the association between tuberculosis rates and housing conditions in the wards of Edinburgh. Studies in other countries point to similar conclusions; Copenhagen data for 1865 to 1874, Hamburg records from 1896 to 1900, Buffalo data from the 1940 Census (Terris, 1948), and many other analyses consistently disclose the influence of socio-economic circumstances on tuberculosis. But the relative importance of individual constituents in the "social complex" has not been determined, and the interaction of economic and social factors is such that components of great importance in one country may, in another, in the presence of exceptional local conditions, have a comparatively minor influence. The present study of Glasgow tuberculosis in the period 1930–47 aims at separating the influence of certain social factors as they occurred, and ascertaining the relative importance of their contributions. As a result it appears that, in Glasgow, housing conditions form the largest single factor in the "complex". This may seem remarkable, but in Scotland, and particularly in Glasgow (see Appendix B.1), housing is of such an unusual character as to constitute an exceptional local circumstance. Elsewhere a lesser association between disease and housing may be found, but in Glasgow the association is of an unusual nature.

## Trends of Respiratory Tuberculosis in Glasgow

The mortality and notification rates in Glasgow are among the highest in Great Britain, and for the last 100 years have been considerably higher than the Scottish averages (Fig. 1, opposite).

Nevertheless, there was a considerable decline in Glasgow mortality throughout the late nineteenth and early twentieth centuries. In 1908 this improvement was halted, but the rate again declined steadily during and after World War I except for the period of the 1920–23 depression. In 1926 the lowest death rate so far recorded, less than 8·8 per 10,000, was reached. But from then onwards there was no marked change; downward fluctuations during the 1930–34 depression were followed by slight increases from 1934 until 1939. In 1940 the death rate rose from 8·6 to 11·3; it continued at this high level, and in 1948 reached 11·4, the highest rate for 30 years.

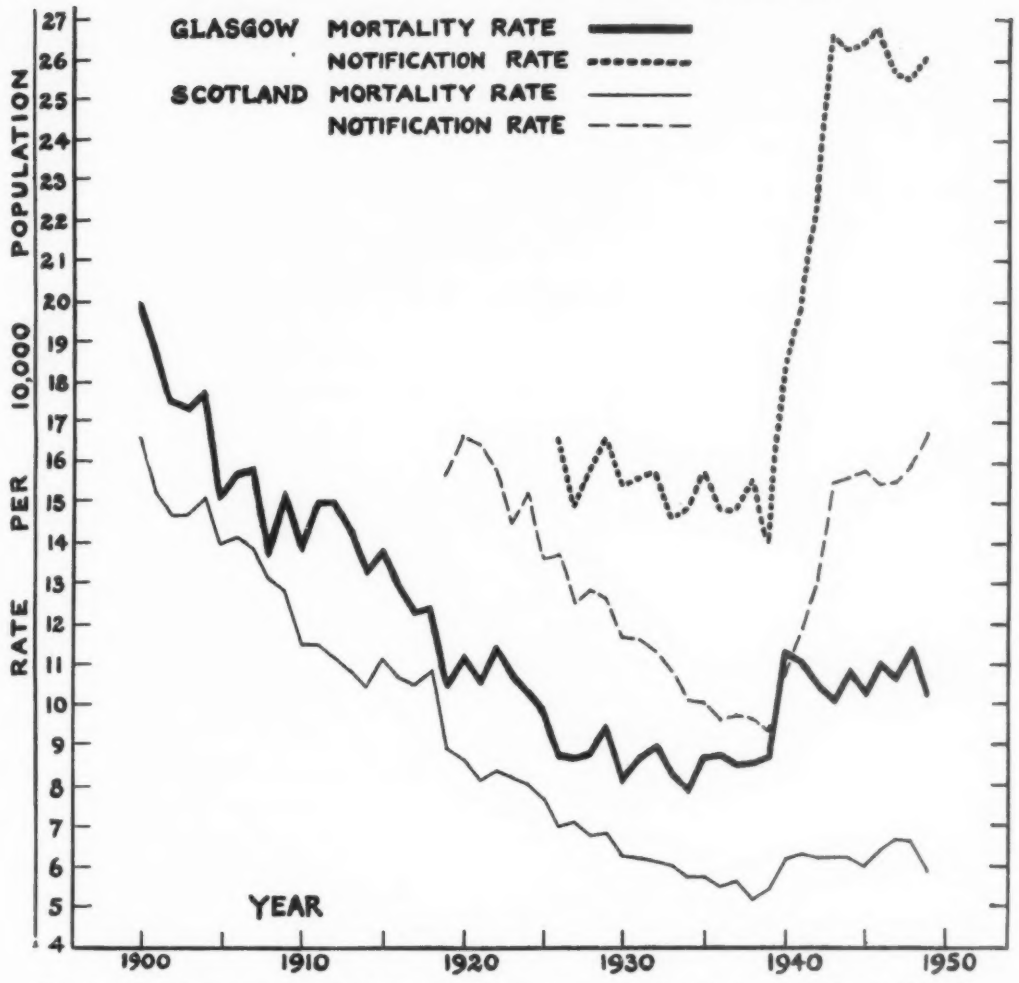



FIG. 1.—Respiratory tuberculosis rates in Glasgow, and in the whole of Scotland, 1900–50.

In 1949, and again in 1950, there was a considerable decline, which has been carried further in the first half of 1951. Yet Glasgow mortality is still higher than the Scottish average, and is above the rate of other large towns (Table I, overleaf).

When compared with the trends in cities of England and Wales, the contrast is more marked, for until the early 1930s the Glasgow rates were lower than those in many industrial areas. But in Liverpool, Manchester, Newcastle, and Cardiff, where the mortality rates at the beginning of the 1930s were 10 or more, the earlier decline continued until 1939. And though in these towns the rates rose sharply in the initial years of war, the decline soon reappeared; and the rates have fallen markedly since 1945. Thus the Glasgow rate, in spite of the great improvements in the last two years, is still the highest of the larger cities of Great Britain.

A study of the notifications of new cases each year similarly reveals that from 1930–39 there was little improvement; and a comparison with other localities again

TABLE I

RESPIRATORY TUBERCULOSIS MORTALITY, 1932–49, IN THE LARGEST CITIES OF SCOTLAND, ENGLAND AND WALES. Crude mortality rate per 10,000 population.

| Year .. | 1932 | 1933 | 1934 | 1937 | 1938 | 1939 | 1945 | 1946 | 1947 | 1948 | 1949 | 1950 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Glasgow .. | 8·9 | 8·2 | 7·9 | 8·5 | 8·5 | 8·6 | 10·3 | 11·0 | 10·7 | 11·4 | 10·1 | 8·5 |
| Edinburgh .. | 7·0 | 7·1 | 6·6 | 6·4 | 6·1 | 6·0 | 5·1 | 6·4 | 6·5 | 6·5 | 5·5 | 4·9 |
| Dundee .. | 6·1 | 5·8 | 5·4 | 5·7 | 6·2 | 6·1 | 6·8 | 7·0 | 8·2 | 6·5 | 7·5 | 5·9 |
| Aberdeen .. | 4·7 | 5·4 | 5·2 | 4·0 | 3·8 | 3·8 | 4·3 | 4·0 | 3·5 | 3·2 | 3·1 | 2·1 |
| London .. | 8·0 | 8·0 | 7·4 | 6·9 | 6·2 | 6·9 | 7·8 | 6·2 | 6·3 | 5·7 | 4·7 | |
| Birmingham | 8·4 | 8·6 | 7·2 | 7·3 | 6·9 | 7·7 | 6·6 | 5·8 | 6·2 | 5·7 | 5·3 | |
| Liverpool .. | 11·3 | 11·8 | 10·1 | 8·4 | 8·1 | 8·0 | 8·9 | 7·8 | 7·8 | 7·8 | 6·8 | |
| Manchester.. | 10·0 | 10·4 | 10·1 | 9·2 | 8·6 | 8·6 | 8·0 | 6·9 | 6·6 | 6·9 | 6·0 | |
| Newcastle .. | 9·8 | 9·2 | 9·8 | 9·4 | 8·6 | 8·3 | 8·3 | 7·8 | 8·8 | 7·4 | 7·6 | |
| Cardiff .. | 10·4 | 10·3 | 9·1 | 8·0 | 8·4 | 8·7 | 8·2 | 7·3 | 7·0 | 6·8 | 6·4 | |

demonstrates the divergence of the Glasgow trend. In the early 1930s the notification rate in Glasgow was higher than that in Scotland as a whole, and throughout the decade, whilst the Scottish rate fell progressively, the Glasgow rate continued at its high level. In England and Wales the notification rate decreased greatly, and though it increased sharply in the early years of war the decline was soon resumed. But in Glasgow (as in the rest of Scotland) notifications mounted continuously during the war, and remained at these higher levels after the war. New notifications in Glasgow averaged 15 per 10,000 persons in the 1930s; in recent years they have averaged 26 to 27 per 10,000 persons. In 1949 the number of new cases recorded was the highest since 1910, and the Medical Officer of Health for Glasgow (1949a), reporting on

TAB

RESPIRATORY TUBERCULOSI ASGOW V

Rates of Mortality (M) and Notifications (N) 000 War

| Year .. .. .. .. | 1930–1 | 1931–2 | 1932–3 | 1933–4 | 1934–5 | 1935–6 | 1936–7 |
|---|---|---|---|---|---|---|---|
| *Mortality Rate:* M | | | | | | | |
| Range .. .. .. .. | 3·66–13·50 | 2·60–12·45 | 2·24–12·59 | 2·66–11·55 | 3·08–13·36 | 3·47–15· | 41-14·4 |
| Mean Rate .. .. .. | 7·98 | 8·29 | 8·24 | 7·77 | 7·89 | 8·23 | 8·20 |
| Standard Deviation of Mean | 2·66 | 2·46 | 2·61 | 2·34 | 2·67 | 2·99 | 2·64 |
| Coefficient of Variation .. | 33·38% | 29·63% | 31·62% | 30·09% | 33·81% | 36·29% | 32·23% |
| *Log. of Mortality:* L .. .. | | | | | | | |
| Mean Log. Rate .. .. | 0·876 | 0·894 | 0·888 | 0·868 | 0·870 | 0·884 | 0·887 |
| Standard Deviation of Mean | 0·156 | 0·160 | 0·172 | 0·150 | 0·159 | 0·173 | 0·166 |
| Coefficient of Variation .. | 17·75% | 17·93% | 19·38% | 17·32% | 18·27% | 19·52% | 18·69% |
| *Notification Rate:* N | | | | | | | |
| Range .. .. .. .. | 6·46–25·22 | 5·64–24·18 | 4·88–21·12 | 5·50–20·95 | 5·75–20·64 | 6·55–22· | 89-21·4 |
| Mean Rate .. .. .. | 14·73 | 14·79 | 14·26 | 13·82 | 14·33 | 14·29 | 14·04 |
| Standard Deviation of Mean | 4·62 | 4·34 | 3·82 | 3·85 | 4·20 | 4·72 | 4·62 |
| Coefficient of Variation .. | 31·34% | 29·37% | 26·80% | 27·86% | 29·27% | 33·01% | 32·94% |

hospital accommodation and general deficiencies, wrote:

> Of these the waiting list for pulmonary tuberculosis is the largest, and the position both from the point of view of the patients and of the community is most grave. . . . Glasgow to-day has over 12,000 tubercular persons on the registers of this Department, this number being 3,000 more than the figure prior to the war.

One of the most disturbing features of the tuberculosis position in Glasgow (and indeed in Scotland) is the fact that the increases in new cases have particularly affected the younger age groups. Actual notifications in these vulnerable groups are shown in Fig. 2 (overleaf). Since 1945 the rate for young women aged 15–24 has been nearly 65 new cases per 10,000; and for young men of similar age the annual rate of new notifications has until recently been well above 40 per 10,000. Moreover, although the crude mortality rate has fallen markedly since 1948, the specific mortality rates for certain age–sex groups have not declined, and the rates for young women are considerably above the levels of 20 years ago.

The rates for Glasgow as a whole were, of course, exceeded in many of the wards (Table II). Throughout the 1930s the annual averages of the ward rates of mortality and notifications wavered about 8 and 14·5 per 10,000 respectively; but at least twelve of the larger wards had rates from 25 to 75 per cent. higher than these averages. Since the war, the mean of the ward mortality rates has been well above 10 per 10,000, and that of the notification rates above 25 per 10,000; but in the larger wards mortality ranged from 12 to 18 per 10,000, whilst in seven wards notifications were between 33 and 38 per 10,000 each year.

The Edinburgh ward rates for the 1930s presented a different picture. Some decline was apparent in both mortality and notification rates, the mean of mortality rates falling from 7·5 to 6·3 and of notification rates from 12·3 to 10·6. Thus the differences between the Edinburgh and Glasgow averages were consistent though small in the

TAB

RCULOSI ASGOW WARDS, 1930–47

ons (N) 00 Ward Population (2-year moving averages.).

| 1935-6 | 1936-7 | 1937–8 | 1938–9 | 1939–40 | 1940–1 | 1944–5 | 1945–6 | 1946–7 |
|---|---|---|---|---|---|---|---|---|
| ·47–15·7 | ·41–14·47 | 1·74–12·76 | 2·46–16·10 | 2·57–15·23 | 3·11–16·80 | 3·10–16·91 | 2·69–16·18 | 3·57–18·41 |
| 8·23 | 8·20 | 8·17 | 8·32 | 9·61 | 10·72 | 10·34 | 10·24 | 10·57 |
| 2·99 | 2·64 | 2·77 | 2·92 | 3·09 | 3·17 | 3·25 | 3·30 | 3·06 |
| 36·29% | 32·23% | 33·97% | 35·10% | 32·20% | 29·54% | 31·45% | 32·23% | 28·96% |
| 0·884 | 0·887 | 0·879 | 0·889 | 0·955 | 1·007 | 0·989 | 0·983 | 1·003 |
| 0·173 | 0·166 | 0·188 | 0·177 | 0·169 | 0·155 | 0·161 | 0·169 | 0·145 |
| 19·52% | 18·69% | 21·35% | 19·87% | 17·65% | 15·40% | 16·32% | 17·19% | 14·45% |
| 55–22·5 | ·89–21·44 | 5·76–23·04 | 5·86–23·89 | 6·88–27·19 | 8·69–27·93 | 10·27–38·18 | 12·28–35·62 | 11·80–38·18 |
| 14·29 | 14·04 | 14·64 | 14·42 | 15·73 | 18·56 | 25·21 | 25·46 | 25·70 |
| 4·72 | 4·62 | 4·66 | 4·24 | 4·66 | 5·48 | 6·55 | 6·48 | 6·70 |
| 33·01% | 32·94% | 31·84% | 29·42% | 29·64% | 29·53% | 25·97% | 25·47% | 26·06% |

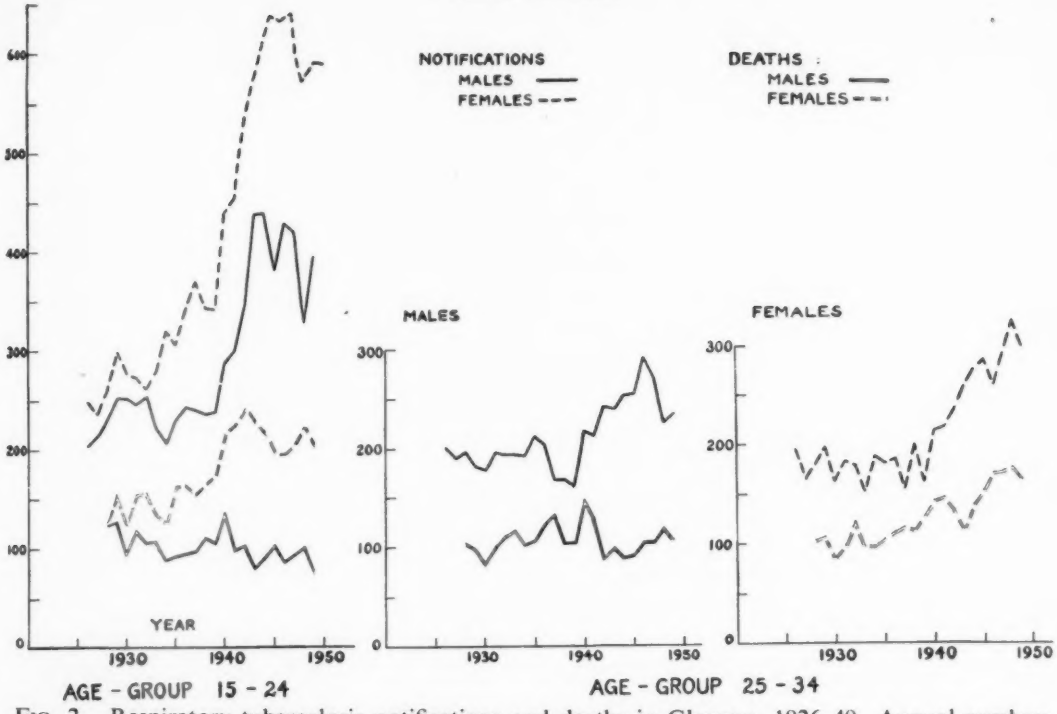



FIG. 2.—Respiratory tuberculosis notifications and deaths in Glasgow, 1926–49. Annual numbers for certain age-sex groups.

early 1930s. But the divergence increased gradually, and just before the war the differences were significant.

It appears, then, that in Glasgow the trends of respiratory tuberculosis have not been similar to those in towns of England and Wales, nor to those in other Scottish cities. In the last 20 years particularly, the movements of the Glasgow rates have exhibited a highly individual character which requires to be more fully examined.

## SCOPE OF THIS STUDY

The main purpose of this study is to investigate the influence of socio-economic conditions in Glasgow on tuberculosis rates from 1930 to 1947, and to break down the "social complex" into components the relative importance of which can be evaluated. It must also be considered whether the contributions of certain social factors vary at different times or in different cities; and whether the findings of the Edinburgh study (Stein, 1950), which demonstrated a strong association between housing and tuberculosis, are confirmed. Finally, the relationships between respiratory tuberculosis and the social complex are compared with those exhibited by other diseases.

This investigation has concentrated on the smallest administrative units for which records are available, the wards of Glasgow. The smaller and more homogeneous the areas considered, the more genuinely do social or economic indices represent characteristics of real meaning in the locality. Besides the main investigation of the 38 wards, some analyses have also been made of tuberculosis in the ten Employment

Exchange Areas of Glasgow; these areas are larger and more heterogeneous, and the results of these analyses are less clear-cut.

The present study deals firstly with the relationships of several social variables and the incidence and mortality rates of respiratory tuberculosis from 1930 to 1947. General crowding in houses, additional overcrowding defined by statutory standards, unemployment, and poverty are discussed. The results are also compared with previous analyses of Edinburgh data, to find whether the association between disease and housing conditions is of a similar order in the two cities.

For the same period, 1930–47, the relationship between respiratory tuberculosis mortality and previous notification is examined; and some confirmation of Edinburgh findings is given.

The two decades investigated have been remarkable not only for the increases in tuberculosis but also for the frequency and variety of social and economic disturbances. A detailed examination is therefore made of tuberculosis mortality and notification in the pre-war and post-war periods, and of the increases which occurred in the interval. The increases in the tuberculosis rates in individual wards are examined to see whether they are related to pre-war environmental circumstances in those wards.

In the first place the direct relationship between each disease rate and each social variable is examined, but since this does not take into account the interaction of various social factors, the influence of social variables operating simultaneously is also examined, and thus a measure of the relative importance of the contribution of each is obtained. The complex relationships between respiratory tuberculosis and the combined distributions of ordinary crowding, overcrowding, unemployment, and poverty, show that the major portion of the ward differences in tuberculosis corresponds to the ward divergencies in this "complex".

Additional analyses are included of the associations of these social factors with non-respiratory tuberculosis mortality and incidence, with infant mortality, and with total mortality from all causes in the period 1930–47. Comparisons between these associations and the relationships found for respiratory tuberculosis indicate the differing importance of different social factors. The social distributions themselves are also examined in detail, and the inter-relations between ordinary crowding, overcrowding, poverty, and unemployment, at different periods of the two decades, are analysed.

It would have been desirable to consider also other factors commonly believed to be relevant to the incidence of tuberculosis. But the dearth of recorded data for small definable areas hampers precise investigation, and this study has been restricted to material for which reliable and continuous data were available.

### Statistical Methods

The methods used throughout the following analyses of relationships between two or more distributions are those of correlation and regression analysis; and the importance of any relationship is assessed by analysis of variance, and by consideration of the proportions of "explained" variance, and of residual "unexplained" variance, in the different regressions.

In the descriptions of each separate distribution, whether of a disease or of a social index, the

ranges, means, and standard deviations of the means are given. The limits of the ranges and means show the time-trends of the rates in general and in the worst or best wards. The standard deviations of the means show the extent to which the individual ward rates are scattered about the means. The time-trends of these dispersions are indicated by the coefficients of variation, measures of dispersion which relate each standard deviation to the appropriate mean. In the various distributions of disease rates and social conditions, the scale of the divergencies between the wards may be compared by direct comparison of these coefficients of variation.

In the single relationship between a disease variable and a social variable, direct correlation coefficients and simple regression coefficients and equations are calculated, and the proportions of "explained" variance and residual variance "unexplained" are shown. In the case of mortality relationships, curvilinear regression equations are used.

Correlation coefficients measure the simultaneous variations of the two distributions, and thus indicate the significance or otherwise of the association investigated.

Regression coefficients measure the estimated response of the disease variable to a unit change in the independent social variable, and thus indicate the differences in numbers of deaths (or cases) associated with social differences in wards.

"Explained" variance measures the extent to which the estimate represented by the regression equation agrees with the mortality (or notification) rates actually observed; thus it indicates the magnitude of the contribution of that social factor to mortality (or incidence). Further, the ratio of "explained" variance to residual variance shows the significance and reliability of the estimate.

This is, in the main, the standard procedure generally adopted to ascertain the importance of relationships between disease rates and external factors (though frequently only correlation coefficients are examined). It is a reliable method on the whole, provided that the units in the distributions so brought into relation do genuinely represent homogeneous groups. In general, the higher the correlation, the more important is the relationship found, and the greater is the influence of that factor on the disease. But if the social factor represented by the independent variable were itself partly dependent on some other important social condition, it might appear to have a greater influence, in connexion with the disease, than is actually attributable to it alone. A high correlation coefficient need not always indicate a high degree of *direct* association between the two specified distributions, but may express the strong influence of the whole social complex on disease. A rigid analysis has to separate the explicit effect of one social variable from the effects implicit in its interrelation with other social factors; it has to examine a compound relationship in which the most important factors are introduced together and their interaction is taken into account.

For this reason, a considerable part of the investigation carried out in the first place by direct correlation and simple regression analysis is repeated and enlarged by multiple correlation and multiple regression analysis. The analysis of variance in the multiple regression, the proportion of the variance explained, and the separation of the latter into the components contributed by the different social variables, together give a better picture of the compound relationship and a more precise estimate of each factor's importance.

The duplication entailed in successive multiple analyses of relationships already investigated in the preliminary simple regressions and direct correlations was undertaken for two purposes. Firstly, in the Edinburgh study the only social indices used were those for ordinary crowding and overcrowding; it is therefore desirable to show that the strong associations found in those analyses were indeed direct associations of high order, and *not* the implicit effects of some other influence. Secondly, the recapitulation by multiple regressions discloses that some of the high direct correlations in simple regressions are due to the indirect effect of inter-relation between the social factors; the multiple relationships reveal which of the social variables are dominant factors. Although direct correlations in simple regressions do, on the whole, show the order of importance of the various social indices, they do not give reliable estimates of the magnitudes of the respective contributions. The recapitulation by multiple association exposes the possible weakness of a conclusion based only on a direct correlation coefficient.

In addition, the complex equations disclose the partial correlations between the disease variable and each component of the social complex. The residual intercorrelations between the component social factors are also revealed.

Standard errors of correlation and regression coefficients are tabled, and significance can be assessed by the ratio of a statistic to its standard error; if this ratio be greater than 2, the statistic is usually held to be significant, since the likelihood of such a value arising by pure chance is less than

1 in 20. In this study, however, the more rigorous "$F$" test* of the ratio of "explained" variance to residual variance is used to judge the level of significance of any relationship expressed as a regression. For significance levels of partial correlation and regression coefficients, the "$t$" test† is applied.

In addition to the tables of data and results of the analyses outlined above, diagrams illustrate the closeness and consistency of the associations found. Scatter diagrams, with regression lines or curves fitted, indicate the progressive increase in disease rates as social conditions in the wards deteriorate, and show the nearness of the actual ward rates to the regression estimates. Variance analysis diagrams, showing the relationships involving each social factor singly and in combination, also demonstrate the small proportion of the variance unaccounted for by the complex relationships.

## I. Respiratory Tuberculosis in Relation to Different Social Factors

The separate relationships between each mortality or notification distribution and each social variable from 1930 to 1947 are summarized in Table III (overleaf, pp. 10–11); coefficients of correlation and regression are given, and the variance "explained" by each regression is shown. These results demonstrate that each social factor was closely and continuously associated with ward differences in respiratory tuberculosis. Correlation coefficients, regressions, and "explained" variance all show the high level of significance of each relationship. But the closeness of the connection between the distribution of mortality or notifications and the various social distributions was not the same. Certain social factors were more strongly associated with the distribution of mortality than with that of notification; and the influence of some social factors became more important in the post-war years.

The most consistent and largest associations of tuberculosis mortality were those with ordinary crowding, and the correspondence between these two distributions was even more marked in the post-war period. Overcrowding was highly correlated with mortality in the early 1930s, but the association lessened slightly in later years. Unemployment was less markedly associated with mortality than was ordinary crowding or overcrowding; nevertheless, the connexion with unemployment was highly significant at all times. Equally, the distribution of poverty corresponded most significantly with ward differences in mortality, and this correspondence became even closer after 1945.

The correlations between ward notification rates and the various social distributions differed only a little from those described for mortality, and all were highly significant. The distribution of ordinary crowding corresponded closely with that of notifications, but this was not so marked as with mortality; notification rates in the early 1930s were as closely associated with overcrowding and unemployment as with ordinary crowding. In the later '30s the connexion between notifications and unemployment increased steadily, whilst the influence of overcrowding became still greater. Poverty in the district, on the other hand, was not markedly associated with ward notification rates before 1939, correlations being rather lower than in the case of mortality; in the post-war period, notifications corresponded more markedly with the distribution of poverty than with any other social factor.

*Fisher (1925) first introduced the statistic $z$, which relates to two different estimates of variance, and published tables of the $z$ distribution. From these Snedecor (1934) extracted a table for the variance ratio, and he named this ratio "$F$" after Fisher.

†The statistic "$t$" represents the ratio of a deviation to its estimated standard error.

TABLE

RESPIRATORY TUBERCULOSIS IN RELATION TO SOCIAL FACTORS IN INDIVIDUAL WARDS

Correlations, Regressions, and Analyses of Variance of TUBERCULOSIS MORTALITY and NOTIFICATIONS,

M: MORTALITY Rate per 10,000 Ward Population.
L: Log. of Mortality Rate.
N: NOTIFICATION Rate per 10,000 Ward Population.
A: ORDINARY CROWDING—density of dwelling-occupation (average persons per house in each ward).
U: UNEMPLOYMENT—adult males registered as unemployed in relation to total population (males unemployed per cent. of ward population).
P: OVERCROWDING—density of room-occupation (average persons per room in each ward).
T: Excess room-crowding of population (percentage of ward population living 2+ per room).
O: Statutory overcrowding (percentage of houses found to be overcrowded).

RESPIRATORY TUBERCULOSIS MORTALITY and NOTIFICATIONS in relation to ORDINARY CROWDING

| Ordinary Crowding: A | 1930 | 1931 | 1932 | 1933 | 1934 | 1935 | 1936 | 1937 | 1938 | 1939 | 1940 |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Mortality Rate: M | 1930–1 | 1931–2 | 1932–3 | 1933–4 | 1934–5 | 1935–6 | 1936–7 | 1937–8 | 1938–9 | 1939–40 | 1940–1 |
| Correlation Coefficient *r* .. | ·689 | ·747 | ·657 | ·713 | ·665 | ·620 | ·760 | ·804 | ·760 | ·644 | ·709 |
| Regression Coefficient *b* .. | 6·74 | 6·27 | 6·74 | 6·64 | 7·06 | 6·30 | 6·82 | 7·73 | 7·72 | 7·00 | 8·47 |
| Standard Error of *b* .. | 1·17 | 0·96 | 1·31 | 1·09 | 1·32 | 1·33 | 0·97 | 0·91 | 1·10 | 1·38 | 1·40 |
| *Log. of Mortality:* L | | | | | | | | | | | |
| Correlation Coefficient *r* .. | ·723 | ·770 | ·633 | ·699 | ·694 | ·661 | ·766 | ·820 | ·812 | ·675 | ·730 |
| Regression Coefficient *b* .. | ·444 | ·416 | ·429 | ·418 | ·439 | ·387 | ·449 | ·533 | ·499 | ·400 | ·427 |
| Standard Error of *b* .. | ·071 | ·058 | ·089 | ·071 | ·076 | ·073 | ·063 | ·062 | ·060 | ·073 | ·067 |
| "Explained" Variance .. | 52% | 59% | 40% | 49% | 48% | 44% | 59% | 67% | 66% | 46% | 53% |
| Variance Ratio F .. .. | 38·3 | 51·0 | 23·4 | 34·4 | 33·5 | 27·9 | 51·1 | 73·7 | 69·7 | 30·1 | 41·1 |
| *Notification Rate:* N | | | | | | | | | | | |
| Correlation Coefficient *r* .. | ·626 | ·708 | ·706 | ·655 | ·666 | ·610 | ·680 | ·769 | ·746 | ·687 | ·741 |
| Regression Coefficient *b* .. | 12·43 | 10·36 | 10·64 | 10·02 | 11·11 | 9·76 | 10·67 | 12·43 | 11·02 | 11·25 | 15·32 |
| Standard Error of *b* .. | 2·61 | 1·75 | 1·80 | 1·93 | 2·07 | 2·12 | 1·92 | 1·72 | 1·64 | 1·98 | 2·32 |
| "Explained" Variance .. | 39% | 50% | 50% | 43% | 44% | 37% | 46% | 59% | 56% | 47% | 55% |
| Variance Ratio F .. .. | 22·6 | 35·2 | 34·9 | 27·0 | 28·7 | 21·3 | 30·9 | 52·2 | 45·2 | 32·2 | 43·8 |

RESPIRATORY TUBERCULOSIS MORTALITY and NOTIFICATIONS in relation to UNEMPLOYMENT

| Unemployment Rate: U | 1930 | 1931 | 1932 | 1933 | 1934 | | 1936 | 1937 | 1938 | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Mortality Rate: M | 1930–1 | 1931–2 | 1932–3 | 1933–4 | 1934–5 | | 1936–7 | 1937–8 | 1938–9 | | |
| Correlation Coefficient *r* | ·751 | ·691 | ·610 | ·683 | ·656 | | ·671 | ·622 | ·588 | | |
| Regression Coefficient *b* .. | ·806 | ·473 | ·397 | ·426 | ·452 | | ·536 | ·571 | ·701 | | |
| Standard Error of *b* .. | ·120 | ·084 | ·087 | ·076 | ·087 | | ·099 | ·120 | ·161 | | |
| *Log. of Mortality:* L | | | | | | | | | | | |
| Correlation Coefficient *r* .. | ·757 | ·679 | ·609 | ·686 | ·655 | | ·663 | ·643 | ·602 | | |
| Regression Coefficient *b* .. | ·047 | ·030 | ·026 | ·028 | ·031 | | ·035 | ·037 | ·043 | | |
| Standard Error of *b* .. | ·007 | ·006 | ·006 | ·005 | ·006 | | ·007 | ·007 | ·010 | | |
| "Explained" Variance .. | 57% | 46% | 37% | 47% | 43% | | 44% | 41% | 36% | | |
| Variance Ratio F .. .. | 45·1 | 29·9 | 20·6 | 32·0 | 27·1 | | 28·2 | 25·5 | 20·4 | | |
| *Notification Rate:* N | | | | | | | | | | | |
| Correlation Coefficient *r* .. | ·586 | ·644 | ·731 | ·724 | ·741 | | ·763 | ·734 | ·650 | | |
| Regression Coefficient *b* .. | 1·090 | ·779 | ·698 | ·742 | ·894 | | 1·003 | 1·140 | 1·126 | | |
| Standard Error of *b* .. | ·255 | ·157 | ·110 | ·118 | ·135 | | ·142 | ·176 | ·220 | | |
| "Explained" Variance .. | 34% | 41% | 54% | 52% | 55% | | 58% | 54% | 42% | | |
| Variance Ratio F .. .. | 18·3 | 24·8 | 40·3 | 39·6 | 43·8 | | 50·1 | 42·0 | 26·3 | | |

CORRELATION COEFFICIENTS: The likelihood that a correlation coefficient of this magnitude would arise by chance is { less than 1 in 1000 if *r* is greater than ·512 / less than 1 in 100 if *r* is greater than ·413

Hence all these results are

III

FACTORS IN WARDS OF GLASGOW, 1930–1947

in individual relationships with ORDINARY CROWDING, OVERCROWDING, UNEMPLOYMENT, and POVERTY.

V: POVERTY—valuation of district (proportion of houses of high rentals in relation to other rentals in each ward).
R: Poor relief according to Outdoor Rolls of Welfare Department (percentage of ward population chargeable to Welfare Department).
Correlation Coefficient *r*: in individual relationship between distribution of disease rate and distribution of social condition.
Regression Coefficient *b*: in individual relationship, average difference in disease rate associated with a unit increase in social index.
Standard Error of *b*: extent of variation of *b* which might be expected.
"Explained" Variance, %: proportion of variance in disease rate associated with ward differences in social conditions.
Variance Ratio *F*: significance level of relationship between disease distribution and distribution of social condition.

| | | | RESPIRATORY TUBERCULOSIS MORTALITY and NOTIFICATIONS in relation to OVERCROWDING | | | | | |
|---|---|---|---|---|---|---|---|---|
| 1944 | 1945 | 1946 | Index P: 1931 (Persons per room) | | Index T: 1931 (Percentage living 2+ per room) | | Index O: 1935 (Percentage houses overcrowded) | |
| 1944–5 | 1945–6 | 1946–7 | 1930–1 | 1931–2 | 1930–1 | 1931–2 | 1934–5 | 1935–6 |
| ·775 | ·832 | ·748 | ·811 | ·869 | ·828 | ·839 | ·687 | ·706 |
| 9·60 | 10·37 | 8·54 | 4·77 | 4·88 | ·122 | ·118 | ·131 | ·151 |
| 1·31 | 1·15 | 1·26 | ·581 | ·463 | ·014 | ·013 | ·023 | ·025 |
| ·813 | ·861 | ·803 | ·849 | ·884 | ·872 | ·823 | ·728 | ·742 |
| ·500 | ·550 | ·484 | ·292 | ·313 | ·0075 | ·0065 | ·0083 | ·0091 |
| ·060 | ·054 | ·053 | ·031 | ·028 | ·0007 | ·0007 | ·0013 | ·0014 |
| 66% | 74% | 65% | 72% | 78% | 76% | 68% | 53% | 55% |
| 70·2 | 103·1 | 82·3 | 90·2 | 125·2 | 111·0 | 75·8 | 40·7 | 44·0 |
| ·752 | ·733 | ·728 | ·650 | ·701 | ·712 | ·760 | ·824 | ·783 |
| 18·75 | 17·96 | 18·16 | 6·62 | 6·73 | ·182 | ·182 | ·247 | ·264 |
| 2·74 | 2·78 | 2·85 | 1·31 | 1·16 | ·030 | ·026 | ·028 | ·035 |
| 57% | 54% | 53% | 42% | 49% | 51% | 58% | 68% | 61% |
| 46·9 | 41·8 | 40·5 | 25·6 | 33·9 | 36·0 | 47·8 | 76·1 | 57·1 |

| | | | RESPIRATORY TUBERCULOSIS MORTALITY and NOTIFICATIONS in relation to POVERTY | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| 1945 | 1946 | 1947 | Index V (Valuation, 1931) | | | Index V (Valuation, 1936) | | | Index R (Poor Relief, 1945–6) | | |
| 1944–5 | 1945–6 | 1946–7 | 1930–1 | 1931–2 | 1932–3 | 1936–7 | 1937–8 | 1938–9 | 1944–5 | 1945–6 | 1946–7 |
| ·623 | ·632 | ·681 | ·600 | ·721 | ·715 | ·649 | ·656 | ·599 | ·779 | ·735 | ·749 |
| 2·44 | 2·70 | 3·03 | ·154 | ·171 | ·180 | ·166 | ·176 | ·170 | 1·65 | 1·58 | 1·50 |
| ·510 | ·552 | ·543 | ·035 | ·028 | ·030 | ·033 | ·034 | ·038 | ·222 | ·244 | ·221 |
| ·631 | ·641 | ·673 | ·654 | ·788 | ·831 | ·679 | ·767 | ·709 | ·775 | ·723 | ·730 |
| ·119 | ·131 | ·149 | ·0098 | ·0122 | ·0138 | ·0114 | ·0140 | ·0121 | ·0817 | ·0797 | ·0691 |
| ·024 | ·026 | ·027 | ·0019 | ·0016 | ·0016 | ·0021 | ·0019 | ·0020 | ·0111 | ·0127 | ·0108 |
| 40% | 41% | 45% | 43% | 62% | 69% | 46% | 59% | 50% | 60% | 52% | 53% |
| 23·8 | 25·1 | 29·8 | 26·1 | 57·2 | 78·0 | 30·7 | 51·3 | 36·3 | 54·2 | 39·4 | 40·9 |
| ·701 | ·782 | ·753 | ·495 | ·568 | ·675 | ·615 | ·577 | ·500 | ·882 | ·833 | ·805 |
| 6·76 | 7·50 | 7·47 | ·220 | ·238 | ·249 | ·275 | ·261 | ·204 | 3·77 | 3·53 | 3·52 |
| 1·15 | 0·996 | 1·09 | ·065 | ·058 | ·046 | ·059 | ·062 | ·059 | ·336 | ·391 | ·432 |
| 49% | 61% | 57% | 25% | 32% | 46% | 38% | 33% | 25% | 78% | 69% | 65% |
| 34·8 | 56·7 | 47·2 | 11·4 | 16·7 | 29·3 | 21·9 | 17·9 | 11·8 | 126·3 | 81·5 | 66·4 |

VARIANCE RATIO $F$ { "explained" variance / "unexplained" variance }: The probability of chance occurrence is
{less than 0·001 if $F$ is greater than 12·8
{less than 0·01 if $F$ is greater than 7·4
well above 99·9% significance.

These indications of the order of importance of the various social distributions in changing times were also brought out by the regression coefficients and the "explained" variance in the different relationships. Moreover, the regressions confirmed that the associations of the social distributions with mortality were more consistent than with notifications. The upward curve of the regressions in the mortality relationships evidences the steep increases in mortality rates which accompanied the worsening of social conditions. Where crowding or unemployment rates were double those of better wards, tuberculosis mortality was rather more than twice as high.

A fuller examination of correlations and variance absorbed reveals the strength of these associations, with correlations far above the 99·9 per cent. level of significance. Many of the correlation coefficients were as high as 0·75 to 0·85. The proportions of total variance accounted for by regression were in many cases more than 65 per cent. In the post-war period ordinary crowding "accounted for" 70 per cent. of the mortality variance and 55 per cent. of the notifications variance, and unemployment and poverty absorbed similar proportions of the latter. That these relationships cannot be ascribed to chance is clear from the variance ratios: the likelihood of variance ratios of such magnitude arising by chance is less than 0·05 per cent. (*i.e.* less than 1 in 2,000) in all cases. The closeness of the estimated rates of mortality or notifications, as calculated from the regression equations, to the actual rates observed in the wards is demonstrated by the small standard deviation from regression. In most cases, the original standard deviation of the mean is almost halved by the regression.

The coefficients of regression can be translated into actual figures which show the relationship between social inequalities and tuberculosis rates. In pre-war years, a unit difference in density of dwelling-occupation was associated with approximately seven deaths and eleven notifications per 10,000 persons. These divergencies are brought out more sharply by the regression equations, which give the following estimates for notification and death rates according to degree of ordinary crowding in the 1930s:

| Average Density of Dwelling-Occupation in the Ward | Mortality Rate per 10,000 | Notification Rate per 10,000 |
|---|---|---|
| 3 persons per house (below the mean) .. .. | 1 to 2 | 3 to 5 |
| 4 persons per house (approx. the mean) .. .. | 8 to 9 | 14 to 16 |

These striking differences in tuberculosis in very crowded wards became even greater in the post-war period, when a unit difference in ordinary crowding gave an estimated difference of nearly ten deaths and eighteen notifications per 10,000 persons. The equations give the following estimates for 1945–47:

| Average Density of Dwelling-Occupation in the Ward | Mortality Rate per 10,000 | Notification Rate per 10,000 |
|---|---|---|
| 3 persons per house (below the mean) .. .. | 5 to 6 | 15 to 16 |
| 4 persons per house (above the mean) .. .. | 15 | 34 |

These figures evidence the excessive numbers of deaths and cases of tuberculosis in the heavily-occupied wards, and show also that the post-war increases added to the disadvantages of the more crowded wards.

Similarly, regression equations with overcrowding, unemployment, and poverty can be translated into actual numbers. But such translations have less importance in the interpretation of present-day tuberculosis rates, for the lack of post-war data on overcrowding, and the

changes in unemployment levels and in conditions of poverty, make comparisons between the pre-war and post-war periods unreal. During the 1930s, unemployment differences between the wards were associated with gradually increasing differences in mortality and notifications; but these increases were slight, since the actual level of unemployment was declining. In the early 1930s, a difference of 5 per cent. (nearly half the range) in the ward unemployment index was associated with a difference of 2·5 in the death rate and of 4 in the notification rate per 10,000. As unemployment declined, a smaller difference of 3·5 per cent. (again nearly half the range) in the male unemployment percentage gave similar estimates.

Overcrowding differences between the wards, on the other hand, were associated with increasing ward divergencies in tuberculosis in the 1930s. At first an overcrowding difference of 20 per cent. between wards was associated with differences of approximately 2·5 in the death rate and 3·5 in the notification rate per 10,000, but later these differences increased to 3 deaths and more than 5 notifications per 10,000 persons.

The regression equations show the estimated rates of tuberculosis according to percentages overcrowded, and in the years 1935–39 the equations indicate the following rates:

| Percentage Statutorily Overcrowded in the Ward | Mortality Rate per 10,000 | Notification Rate per 10,000 |
|---|---|---|
| Less than 5% .. .. .. .. | 4–4·5 | 7–8 |
| 10% .. .. .. .. .. | 5·5 | 9·5 |
| 30% (approximately the mean) .. .. | 8·5 | 14·5 |
| 50% .. .. .. .. .. | 11·5 | 20 |

These estimates show that in the less overcrowded areas the prevalence of tuberculosis was comparatively low. Poverty regressions also show the varying levels of the disease associated with the distribution of poverty in both pre-war and post-war periods. The post-war regressions indicate the following distribution of tuberculosis according to varying percentages of ward population on relief:

| Percentage on Poor Relief in the Ward | Mortality Rate per 10,000 | Notification Rate per 10,000 |
|---|---|---|
| Less than 1% .. .. .. .. | 6–7 | 15–18 |
| 2% .. .. .. .. .. | 9 | 22 |
| 3% (approximately the mean) .. .. | 10·5 | 25·5 |
| 4% .. .. .. .. .. .. | 12 | 29 |
| 6% .. .. .. .. .. .. | 15 | 36 |

A comparison of the relationships between ordinary crowding or overcrowding and tuberculosis in Glasgow with the findings in Edinburgh (Stein, 1950) reveals a similarity in the estimates and in the time-trends in the two cities. In both sets of regressions, the correlation coefficients are of the same order, and the proportions of "explained" variance are similar. The regression coefficients in the Glasgow relationships are more consistent, and the increases in notifications concomitant with increases in ordinary crowding are greater in Glasgow wards than in Edinburgh wards. On the other hand, the increases in tuberculosis associated with increases in overcrowding seem to be slightly greater in Edinburgh wards. The stronger connexion of Glasgow tuberculosis with ordinary crowding than with overcrowding seems to be again indicated by this comparison with Edinburgh.

Thus the close association between ward differences in respiratory tuberculosis and ward variations in environmental circumstances is revealed by each examination of each relationship. The importance of the "social complex" is manifest, no matter

which index of social conditions be used; yet the relationships of the different social variables to tuberculosis are not by any means identical with each other. Differences in time-trends and differing degrees of importance in relation to mortality or to notifications are apparent in these analyses.

## II. Respiratory Tuberculosis Mortality in Relation to Notification

These analyses of the relationship between the two aspects of the disease were made in order to examine more closely a connexion indicated in the Edinburgh study. Table IV summarizes this relationship between notification rates in any biennial period and mortality in the same and subsequent years in terms of correlation and regression coefficients.

TABLE IV

Respiratory Tuberculosis in Glasgow, 1930–47: Mortality in relation to Notification Rate.
Correlation Coefficients between Notification Rates in any 2-year period and Mortality Rates in the Succeeding Period.

N: Notification Rate per 10,000 Ward Population (2-year moving average)
L: Log. of Mortality Rate per 10,000 Population (2-year moving average)

| Notifications: N | Log. of Mortality: L | Correlation Coefficient: *r* | Standard Error of *r* |
|---|---|---|---|
| 1930–31 | 1931–32 | ·776 | ·0655 |
| 1931–32 | 1932–33 | ·803 | ·0576 |
| 1932–33 | 1933–34 | ·787 | ·0617 |
| 1933–34 | 1934–35 | ·781 | ·0632 |
| 1934–35 | 1935–36 | ·754 | ·0700 |
| 1935–36 | 1936–37 | ·714 | ·0796 |
| 1936–37 | 1937–38 | ·815 | ·0545 |
| 1937–38 | 1938–39 | ·778 | ·0639 |
| 1938–39 | 1939–40 | ·703 | ·0819 |
| 1939–40 | 1940–41 | ·716 | ·0791 |
| 1944–45 | 1945–46 | ·831 | ·0502 |
| 1945–46 | 1946–47 | ·825 | ·0525 |

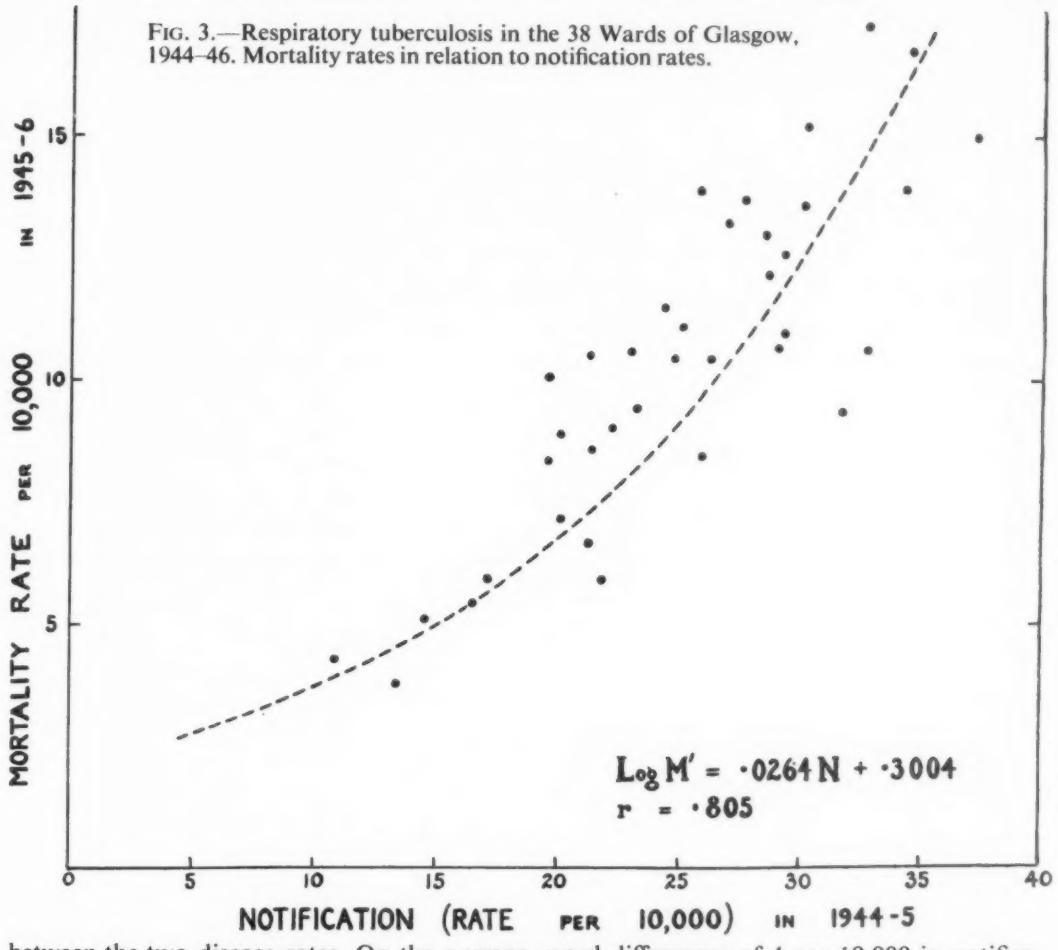
The likelihood that correlation coefficients of this magnitude would arise by chance is less than 1 in 1,000 if *r* exceeds ·512.

The results show, as expected, a close connexion between the two sets of rates. Curvilinear equations were again found better than simple proportional equations in describing the relationship between the two aspects of the disease. Increases in mortality rates became augmented where notification rates were high; in wards where notification rates were twice the mean, mortality rates were rather more than twice the mean. The relationship found in pre-war years in Edinburgh was confirmed; moreover, in Glasgow, the upward curve of mortality, increasing more rapidly than notification rates, was especially marked in the post-war period. Fig. 3 (opposite) illustrates the upward curve of the mortality–notification association.

A more detailed consideration of the correlations and regressions reveals that, during the early 1930s, mortality corresponded closely with the distribution of current notification rates, and was equally strongly associated with notifications in preceding years. But towards the end of the decade the correlations of mortality with preceding notification rates were greater. In the post-war years the highest correlations were found when the interval between notifications and mortality was 2 years.

The regression equations can be translated into figures illustrating the relationship

FIG. 3.—Respiratory tuberculosis in the 38 Wards of Glasgow, 1944–46. Mortality rates in relation to notification rates.




between the two disease rates. On the average, ward differences of 4 per 10,000 in notifications corresponded with differences of 1·5 to 2 per 10,000 in mortality, but the divergencies in mortality were smaller in wards where notification rates were low. The equations give the following estimates for the immediate post-war years.

| Ward Notification Rate per 10,000 | Estimated Mortality Rate per 10,000 |
|---|---|
| 11 to 12 (lowest in the range) | 3·5 to 3·8 |
| 15 to 16 | 4·9 to 5·3 |
| 20 to 21 | 6·9 to 7·5 |
| 25 to 26 (approximately the mean) | 9·5 to 10 |
| 29 to 30 | 12·2 to 12·9 |
| 33 to 34 | 15 to 15·8 |
| 37 to 38 (highest in the range) | 18 to 19 |

Thus in districts in which notification rates were high the population had not only more risk of contracting the disease, but had also a reduced chance of surviving after notification. The fact that this was no less marked in post-war than in pre-war years suggests that this relationship is not an illusory one attributable to lesser accuracy in notification in pre-war years. If pre-war notification practice were the explanation

of this upward curve of mortality in relation to notifications, then the relationship could be expected to diminish in the post-war period.

### III. Increases in Respiratory Tuberculosis between Pre-War and Post-War Years

This investigation of increases in ward tuberculosis rates between 1936–38 and 1945–47 was undertaken to ascertain their relation to ward social conditions at the beginning of the period. The actual and percentage increases in ward mortality and notification rates are shown in Table V (opposite); Table VI (overleaf) summarizes the associations between these increases and each social distribution in 1935–36.

These analyses show that the mean mortality rate increased by 30 per cent. and the mean notification rate by 86 per cent. But many wards experienced greater increases, for both the ranges and variability of the ward increases were high. The regressions disclose that, though the associations between the increased tuberculosis rates in 1945–47 and the social distributions were stronger than before 1939, the actual increases in rates corresponded to a lesser degree with pre-war social conditions in the wards. Moreover, the percentage increases in each ward rate were *negatively* (though not highly) associated with poor social conditions. These consistently negative correlations with each social variable seem to indicate that the better-off wards experienced relatively greater increases in tuberculosis.

A more detailed consideration of these analyses discloses some interesting results. Firstly, the intensified association of the "social complex" with tuberculosis rates in the post-war triennium is apparent in the greater correlations and steeper slopes of the regressions with the various social indices. Regression coefficients and "explained" variance in the relationships of mortality with each social variable were greater in the post-war years. And in the notification equations with the social indices, the post-war increases in regression coefficients were still greater, the "explained" variance in the association with poverty being nearly double that of pre-war years. Fig. 4 (p. 19) shows the ordinary crowding and mortality rates in 1936–38 and in 1945–47, and indicates the regression curve in each relationship. It is evident that the whole level of mortality rates was higher in the post-war period, but in addition the upward curve of the mortality-crowding association was steeper in 1945–47.

Secondly, the actual ward increases in both mortality and notification rates were significantly associated with the distributions of certain social factors. Ward distributions of both unemployment and ordinary crowding in 1935–36 corresponded with increases in the disease rates; these social factors showed low correlations with the ward increases in mortality, and higher correlations with notification increases, significant at the 99 per cent. level. On the other hand, overcrowding and poverty distributions were related not at all to mortality increases, and only slightly to notification increases. Thus it seems that the distribution of increases in the disease was not related to excessively disadvantageous conditions such as statutory overcrowding or recognized poverty, but rather to the ordinary conditions of life in the 1930s, of general crowding and unemployment.

Thirdly (and surprisingly), each of the relationships between the percentage increases in ward tuberculosis rates and the pre-war social distributions revealed small negative correlations. The mortality correlations were in the main quite insignificant, but the negative association between overcrowding and percentage mortality increases was more than 97 per cent. significant. The notification correlations were higher, indicating more than 90 per cent. significance in connexion with ordinary crowding, unemployment, and poverty; and the negative association between overcrowding and percentage increases in notification rates was 99·8 per cent. significant. Although these correlations are small, the uniformly negative

TABLE V

RESPIRATORY TUBERCULOSIS IN PRE-WAR AND POST-WAR TRIENNIA

Mortality, Notifications, and Increases in the 38 Wards of Glasgow in 1936–38 and 1945–47

M : Mortality Rate per 10,000 Ward Population: 3-year average.
L : Log. of Mortality Rate.
N : Notification Rate per 10,000 Ward Population: 3-year average.
$I_M$ : Increase in Mortality—Actual increase in ward rate from 1936–38 to 1945–47.
$C_M$ : Percentage increase in mortality rate.
$I_L$ : Actual increase in log. of mortality rate.
$I_N$ : Increase in Notifications—Actual increase in ward rate from 1936–38 to 1945–47.
$C_N$ : Percentage increase in notification rate.

| | Pre-war Triennium 1936–38 | Post-war Triennium 1945–47 | Interval |
|---|---|---|---|
| *Mortality rate:* M | | | |
| Range of Ward Rates | 2·50 to 13·94 | 2·91 to 15·79 | |
| Mean of Ward Rates | 8·19 | 10·39 | |
| Standard Deviation of Mean | 2·62 | 3·01 | |
| Coefficient of Variation | 32·0% | 29·0% | |
| *Log. of Mortality:* L | | | |
| Mean Log. Rate | 0·887 | 0·994 | |
| Standard Deviation of Mean | 0·166 | 0·153 | |
| Coefficient of Variation | 18·7% | 15·3% | |
| *Notification Rate:* N | | | |
| Range of Ward Rates | 5·45 to 20·56 | 12·78 to 37·57 | |
| Mean of Ward Rates | 14·35 | 25·45 | |
| Standard Deviation of Mean | 4·44 | 6·57 | |
| Coefficient of Variation | 30·9% | 25·8% | |
| *Mortality Increase:* $I_M$ | | | |
| Range of Actual Increases in Ward Rates | | | −2·80 to 5·50 |
| Mean of Actual Increases in Ward Rates | | | 2·19 |
| Standard Deviation of Mean | | | 1·80 |
| Coefficient of Variation | | | 82·1% |
| $I_L$ | | | |
| Mean of Increases in Logs of Mortality Rates | | | 0·107 |
| Standard Deviation of Mean | | | 0·083 |
| Coefficient of Variation | | | 76·8% |
| $C_M$ | | | |
| Range of Percentage Increases in Ward Rates | | | −20·1 to 87·7 |
| Mean of Percentage Increases in Ward Rates | | | 30·3 |
| Standard Deviation of Mean | | | 24·5 |
| Coefficient of Variation | | | 80·9% |
| *Notification Increase:* $I_N$ | | | |
| Range of Actual Increases in Ward Rates | | | 5·76 to 20·36 |
| Mean of Actual Increases in Ward Rates | | | 11·09 |
| Standard Deviation of Mean | | | 3·81 |
| Coefficient of Variation | | | 34·4% |
| $C_N$ | | | |
| Range of Percentage Increases in Ward Rates | | | 37·0 to 249·3 |
| Mean of Percentage Increases in Ward Rates | | | 85·6 |
| Standard Deviation of Mean | | | 43·4 |
| Coefficient of Variation | | | 50·8% |

TABLE VI

RESPIRATORY TUBERCULOSIS IN PRE-WAR AND POST-WAR TRIENNIA
MORTALITY AND NOTIFICATION RATES IN RELATION TO WARD SOCIAL CONDITIONS IN THE 38 WARDS OF GLASGOW, 1936–38 AND 1945–47
INCREASES IN RATES IN RELATION TO PRE-WAR SOCIAL CONDITIONS

| Social Index .. .. | Ordinary Crowding | | Over-crowding | Unemployment | | Poverty | |
|---|---|---|---|---|---|---|---|
| Period .. .. .. | 1936–38 A | 1945–47 A | 1936–38 O | 1936–38 U | 1945–47 U | 1936–38 V | 1945–47 R |
| M: *Mortality Rate* | | | | | | | |
| Correlation Coefficient *r* | ·809 | ·823 | ·772 | ·622 | ·677 | ·640 | ·771 |
| Regression Coefficient *b* | 7·20 | 9·37 | ·144 | ·571 | 2·96 | ·163 | 1·516 |
| Standard Error of *b* .. | ·872 | 1·076 | ·020 | ·120 | ·536 | ·033 | ·209 |
| L: *Log. of Mortality* | | | | | | | |
| Correlation Coefficient *r* | ·840 | ·847 | ·788 | ·644 | ·665 | ·703 | ·742 |
| Regression Coefficient *b* | ·473 | ·488 | ·0093 | ·037 | ·147 | ·011 | ·074 |
| Standard Error of *b* .. | ·051 | ·051 | ·0012 | ·007 | ·028 | ·002 | ·011 |
| "Explained" Variance .. | 71% | 72% | 62% | 42% | 44% | 49% | 55% |
| Variance Ratio F .. | 86·2 | 91·6 | 58·9 | 25·5 | 28·6 | 35·1 | 44·2 |
| N: *Notification Rate* | | | | | | | |
| Correlation Coefficient *r* | ·757 | ·741 | ·823 | ·734 | ·786 | ·596 | ·833 |
| Regression Coefficient *b* | 11·41 | 18·41 | ·261 | 1·14 | 7·50 | ·257 | 3·576 |
| Standard Error of *b* .. | 1·64 | 2·78 | ·030 | ·176 | ·982 | ·058 | ·396 |
| "Explained" Variance .. | 57% | 55% | 68% | 54% | 62% | 36% | 69% |
| Variance Ratio F .. | 48·3 | 43·9 | 75·7 | 42·0 | 58·3 | 19·9 | 81·7 |
| *Mortality Increases*: $I_M$ | | | | | | | |
| Correlation Coefficient *r* | ·207 | | −·018 | ·175 | | ·133 | |
| $C_M$ | | | | | | | |
| Correlation Coefficient *r* | −·155 | | −·356 | −·130 | | −·066 | |
| $C_L$ | | | | | | | |
| Correlation Coefficient *r* | −·366 | | −·467 | −·258 | | −·161 | |
| *Notification Increases*: $I_N$ | | | | | | | |
| Correlation Coefficient *r* | ·407 | | ·253 | ·488 | | ·254 | |
| $C_N$ | | | | | | | |
| Correlation Coefficient *r* | −·294 | | −·484 | −·270 | | −·280 | |

*CORRELATION COEFFICIENTS*

The significance level of a correlation coefficient *r* is:

above 90% (*i.e.* probability of chance occurrence less than 1 in 10) if *r* exceeds ·271
above 95% (*i.e.* probability of chance occurrence less than 1 in 20) if *r* exceeds ·320
above 98% (*i.e.* probability of chance occurrence less than 1 in 50) if *r* exceeds ·376
above 99% (*i.e.* probability of chance occurrence less than 1 in 100) if *r* exceeds ·413
above 99·9% (*i.e.* probability of chance occurrence less than 1 in 1000) if *r* exceeds ·512
above 99·95% (*i.e.* probability of chance occurrence less than 1 in 2000) if *r* exceeds ·566

regressions are not to be ignored. They imply that there was a small but definite correspondence between pre-war social conditions and proportionally greater increases in tuberculosis, particularly in notification rates. Clearly this does not mean that districts with better environments experienced the greater increases in the disease: a connexion between greater *percentage* increases and better pre-war social conditions is easily explicable in view of the all-round increases in tuberculosis. But these negative correlations confirm the fact that the increases *were* all-round, and not confined to one group of people or to a few localities.

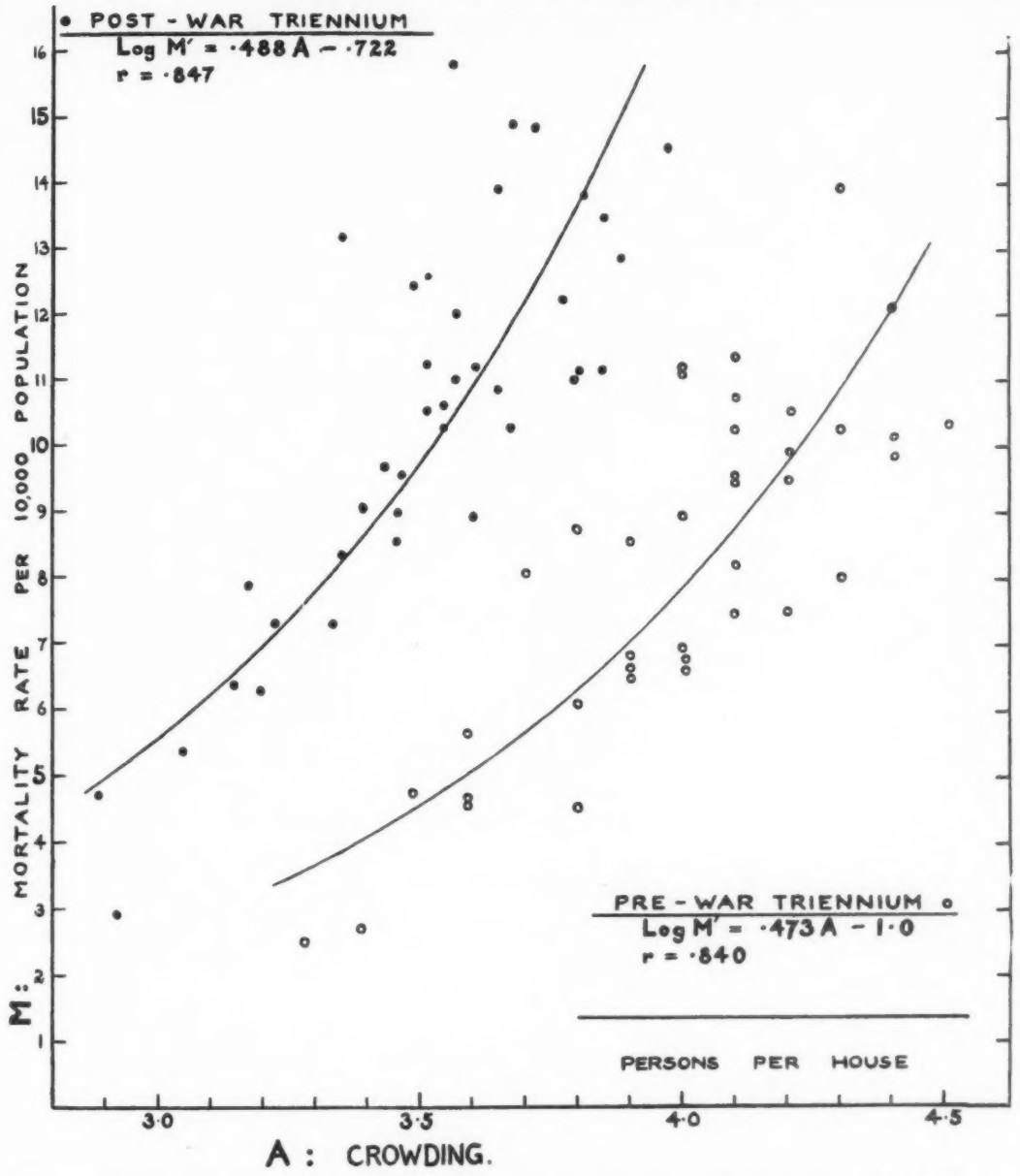



FIG. 4.—Respiratory tuberculosis in the 38 Wards of Glasgow. Mortality rates in relation to ordinary crowding in the pre-war triennium (1936–38), and in the post-war triennium (1945–47).

In general, these analyses indicate that the actual increases in ward tuberculosis rates added greatly to the disadvantageous experience of those districts in which pre-war unemployment and ordinary crowding were disadvantageous and in which pre-war tuberculosis rates were already high. But the widespread increases made a relatively greater mark in wards which in pre-war days had lower disease rates and

better environmental conditions. Such a result is to be expected when one considers that an infectious disease does not confine itself within the worst-housed wards, and that the spread of infection will inevitably be disadvantageous to areas previously least affected.

## IV. Respiratory Tuberculosis and the "Social Complex"

Analyses of the relationships between the distributions of disease rates and the social variables, taken two, three, and four together, have been made for each triennium in the period 1930–47. Since space does not permit the inclusion of all the results, the regressions for the three triennia, 1930–32, 1936–38, and 1945–47, have been selected as representing the whole period. The relationships are summarized in Tables VII, VIII, and IX, in which are set forth partial regression coefficients, partial and total correlations, variance analyses, and significance levels of the associations found between the two aspects of tuberculosis and the many combinations of the social factors. In addition, several regression equations are discussed more fully, and are illustrated by diagrams of variance analyses or of regression estimates.

The findings demonstrate the strong and consistent association between tuberculosis and the group of social factors, and reveal the dominant role of ordinary crowding and overcrowding. Exceptionally large proportions of variance due to ward differences in tuberculosis rates were "explained" by ward divergencies in this complex of social conditions, and the housing factors alone contributed the greater part of this variance. The overlap of the social factors with each other, as shown by their residual inter-correlations was, in most cases, very small. The results confirm that the social factors are not related to notifications in precisely the same manner as to mortality, and that different factors are not of equal weight in the "complex" at all times.

The strength of the connexion between both mortality and notification distributions and the "complex" of social distributions is demonstrated by the high total correlations in the multiple relationships; all were above 0·875 and many were greater than 0·9. Partial correlations with individual social factors within each multiple relationship were also large; and partial regression coefficients of ordinary crowding and overcrowding were highly significant. The variance "explained", both for mortality and for notification distributions, was 80 per cent. or more—a proportion so high that the likelihood of chance occurrence is less than 1 in 2,000 (significant above the 99·95 per cent. level). In these amounts of variance absorbed, ordinary crowding contributed 30 to 50 per cent. in the mortality relationships, and 20 to 30 per cent. in the notification regressions. Overcrowding contributions to the "explained" variance were of similar magnitudes, but in the reverse order in relation to mortality and notifications. In the pre-war regressions poverty and unemployment contributed smaller percentages to "explained" variance; but in the post-war period (in which overcrowding was not considered), the variance "explained" by poverty in association with mortality was nearly 20 per cent. In all periods, the influence of poverty in the mortality relationships was considerable, whereas unemployment played a very small part. But in relation to notifications, poverty contributed little to variance "explained", and unemployment was of greater importance.

The differences in importance of the social factors in association with the two aspects of the disease will be revealed more clearly later in this section, in the analyses of pre-war and

| | SINGLE REGRESSIONS | | | | | DOUBLE REGRESSION with A and T | | | | DOUBLE REGRESSION with A and U | | | | DOUBLE REGRESSION with A and V | | | | DOU |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Relation to | | | | Inter-relations of Social Factors | Partial relation to | | Joint relation to A and T | Inter-relations | Partial relation to | | Joint relation to A and U | Inter-relations | Partial relation to | | Joint relation to A and V | Inter-relations | Partial re |
| | A | T | U | V | | A | T | | | A | U | | | A | V | | | T |
| MORTALITY RATE: M | | | | | | | | | | | | | | | | | | |
| Total Correlation Coefficient | $r_{MA}=\cdot757$ | $r_{MT}=\cdot829$ | $r_{MU}=\cdot691$ | $r_{MV}=\cdot721$ | | | | $R=\cdot910$ | | | | $R=\cdot804$ | | | | $R=\cdot863$ | | |
| Partial Correlation Coefficient $r$ | | | | | $r_{AT}=\cdot731$ | ·587 | ·661 | | | ·604 | ·412 | | | ·603 | ·482 | | | ·781 |
| Regression Coefficient $b$ | $b_{MA}=6\cdot27$ | $b_{MT}=\cdot117$ | $b_{MU}=\cdot473$ | $b_{MV}=\cdot171$ | $r_{AU}=\cdot640$ | 3·82 | ·082 | | | 4·42 | ·240 | | | 4·27 | ·116 | | | ·102 |
| Standard Error of Regression Coefficient | ·914 | ·013 | ·0836 | ·0277 | $r_{AV}=\cdot555$ | ·892 | ·016 | | | ·986 | ·089 | | | ·955 | ·036 | | | ·014 |
| | | | | | $r_{TU}=\cdot702$ | | | | $r_{AT.M}=\cdot211$ | | | | $r_{AU.M}=\cdot246$ | | | | $r_{AV.M}=-\cdot167$ | |
| Original Standard Deviation of Mean of M | 2·457 | 2·457 | 2·457 | 2·457 | $r_{TV}=\cdot763$ | | | 2·457 | | | | 2·457 | | | | 2·457 | | |
| Standard Deviation from Regression | 1·627 | 1·113 | 1·801 | 1·728 | $r_{UV}=\cdot439$ | | | 1·0334 | | | | 1·483 | | | | 1·261 | | |
| "Explained" Variance | 57·4% | 68·8% | 47·8% | 51·9% | | 34·9% | 47·9% | 82·8% | | 40·4% | 24·2% | 64·6% | | 39·1% | 35·3% | 74·4% | | 60·1% |
| Variance Ratio F | 47·1 | 79·1 | 32·0 | 37·8 | | | | 84·2 | | | | 31·9 | | | | 50·9 | | |
| LOG. OF MORTALITY RATE: $^{L}$M | | | | | | | | | | | | | | | | | | |
| Total Correlation Coefficient | $r_{LA}=\cdot770$ | $r_{LT}=\cdot823$ | $r_{LU}=\cdot679$ | $r_{LV}=\cdot788$ | | | | $R=\cdot933$ | | | | $R=\cdot807$ | | | | $R=\cdot884$ | | |
| Partial Correlation Coefficient $r$ | | | | | $r_{AT}=\cdot731$ | ·618 | ·744 | | | ·656 | ·381 | | | ·632 | ·531 | | | ·804 |
| Regression Coefficient $b$ | $b_{LA}=\cdot416$ | $b_{LT}=\cdot0065$ | $b_{LV}=\cdot0303$ | $b_{LV}=\cdot0122$ | $r_{AU}=\cdot640$ | ·257 | ·0049 | | | ·307 | ·014 | | | ·287 | ·0073 | | | ·006 |
| Standard Error of Regression Coefficient | ·058 | ·0007 | ·0055 | ·0016 | $r_{AV}=\cdot555$ | ·054 | ·0007 | | | ·060 | ·006 | | | ·061 | ·0020 | | | ·0008 |
| | | | | | $r_{TU}=\cdot702$ | | | | $r_{AT.L}=\cdot081$ | | | | $r_{AU.L}=\cdot220$ | | | | $r_{AV.L}=-\cdot132$ | |
| Original Standard Deviation of Mean of L | ·160 | ·160 | ·160 | ·160 | $r_{TV}=\cdot763$ | | | ·160 | | | | ·160 | | | | ·160 | | |
| Standard Deviation from Regression | ·104 | ·091 | ·119 | ·100 | $r_{UV}=\cdot439$ | | | ·058 | | | | ·096 | | | | ·076 | | |
| "Explained" Variance | 59·3% | 67·8% | 46·1% | 62·1% | | 36·3% | 50·8% | 87·1% | | 43·8% | 21·4% | 65·2% | | 41·0% | 37·1% | 78·1% | | 63·5% |
| Variance Ratio F | 52·5 | 75·8 | 30·8 | 57·2 | | | | 118·2 | | | | 32·8 | | | | 62·4 | | |
| NOTIFICATION RATE: N | | | | | | | | | | | | | | | | | | |
| Total Correlation Coefficient | $r_{NA}=\cdot708$ | $r_{NT}=\cdot760$ | $r_{NU}=\cdot644$ | $r_{NV}=\cdot568$ | | | | $R=\cdot792$ | | | | $R=\cdot750$ | | | | $R=\cdot743$ | | |
| Partial Correlation Coefficient $r$ | | | | | $r_{AT}=\cdot731$ | ·540 | ·583 | | | 591 | ·351 | | | ·619 | ·304 | | | ·608 |
| Regression Coefficient $b$ | $b_{NA}=10\cdot36$ | $b_{NT}=\cdot182$ | $b_{NU}=\cdot779$ | $b_{NV}=\cdot238$ | $r_{AU}=\cdot640$ | 4·80 | ·125 | | | 7·34 | ·391 | | | 8·91 | ·089 | | | ·133 |
| Standard Error of Regression Coefficient | 1·75 | ·026 | ·156 | ·058 | $r_{AV}=\cdot555$ | 1·27 | ·029 | | | 1·72 | ·176 | | | 1·94 | ·047 | | | ·029 |
| | | | | | $r_{TU}=\cdot702$ | | | | $r_{AT.N}=\cdot320$ | | | | $r_{AU.N}=\cdot321$ | | | | $r_{AV.N}=\cdot028$ | |
| Original Standard Deviation of Mean of N | 4·343 | 4·343 | 4·343 | 4·343 | $r_{TV}=\cdot763$ | | | 4·343 | | | | 4·343 | | | | 4·343 | | |
| Standard Deviation from Regression | 3·111 | 2·864 | 3·371 | 3·624 | $r_{UV}=\cdot439$ | | | 2·728 | | | | 2·913 | | | | 2·948 | | |
| "Explained" Variance | 50·1% | 57·7% | 41·4% | 32·3% | | 23·2% | 39·5% | 62·7% | | 35·5% | 20·8% | 56·3% | | 43·1% | 12·1% | 55·2% | | 42·1% |
| Variance Ratio F | 35·2 | 47·8 | 24·7 | 16·7 | | | | 28·6 | | | | 22·5 | | | | 21·6 | | |

| | SINGLE REGRESSIONS | | | | | DOUBLE REGRESSION with A and O | | | | DOUBLE REGRESSION with A and U | | | | DOUBLE REGRESSION with A and V | | | | D |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Relation to | | | | Inter-relations of Social Factors | Partial relation to | | Joint relation to A and O | Inter-relations | Partial relation to | | Joint relation to A and U | Inter-relations | Partial relation to | | Joint relation to A and V | Inter-relations | Partia |
| | A | O | U | V | | A | O | | | A | U | | | A | V | | | O |
| MORTALITY RATE: M | | | | | | | | | | | | | | | | | | |
| Total Correlation Coefficient | $r_{MA}=\cdot757$ | $r_{MO}=\cdot-0$ | $r_{MU}=\cdot691$ | $r_{MV}=\cdot721$ | | | | $R=\cdot757$ | | | | $R=\cdot804$ | | | | $R=\cdot863$ | | |
| Partial Correlation Coefficient $r$ | | | | | $r_{AO}=\cdot-0$ | ·757 | ·-0 | | | ·604 | ·412 | | | ·603 | ·482 | | | ·781 |
| Regression Coefficient $b$ | $b_{MA}=6\cdot27$ | $b_{MO}=\cdot-0$ | $b_{MU}=\cdot473$ | $b_{MV}=\cdot171$ | $r_{AU}=\cdot640$ | 6·27 | ·-0 | | | 4·42 | ·240 | | | 4·27 | ·116 | | | ·102 |
| Standard Error of Regression Coefficient | ·914 | ·-0 | ·0836 | ·0277 | $r_{AV}=\cdot555$ | ·914 | ·-0 | | | ·986 | ·089 | | | ·955 | ·036 | | | ·014 |
| | | | | | $r_{UO}=\cdot-0$ | | | | $r_{AO.M}=\cdot757$ | | | | $r_{AU.M}=\cdot246$ | | | | $r_{AV.M}=-\cdot167$ | |
| Original Standard Deviation of Mean of M | 2·457 | 2·457 | 2·457 | 2·457 | $r_{UV}=\cdot439$ | | | 2·457 | | | | 2·457 | | | | 2·457 | | |
| Standard Deviation from Regression | 1·627 | 1·627 | 1·801 | 1·728 | | | | 1·627 | | | | 1·483 | | | | 1·261 | | |
| "Explained" Variance | 57·4% | ·-0% | 47·8% | 51·9% | | 57·4% | ·-0% | 57·4% | | 40·4% | 24·2% | 64·6% | | 39·1% | 35·3% | 74·4% | | 60·1% |
| Variance Ratio F | 47·1 | ·-0 | 32·0 | 37·8 | | | | 47·1 | | | | 31·9 | | | | 50·9 | | |
| LOG. OF MORTALITY RATE: $^{L}$M | | | | | | | | | | | | | | | | | | |
| Total Correlation Coefficient | $r_{LA}=\cdot770$ | $r_{LO}=\cdot-0$ | $r_{LU}=\cdot679$ | $r_{LV}=\cdot788$ | | | | $R=\cdot770$ | | | | $R=\cdot807$ | | | | $R=\cdot884$ | | |
| Partial Correlation Coefficient $r$ | | | | | $r_{AO}=\cdot-0$ | ·770 | ·-0 | | | ·656 | ·381 | | | ·632 | ·531 | | | ·804 |
| Regression Coefficient $b$ | $b_{LA}=\cdot416$ | $b_{LO}=\cdot-0$ | $b_{LV}=\cdot0303$ | $b_{LV}=\cdot0122$ | $r_{AU}=\cdot640$ | ·416 | ·-0 | | | ·307 | ·014 | | | ·287 | ·0073 | | | ·006 |
| Standard Error of Regression Coefficient | ·058 | ·-0 | ·0055 | ·0016 | $r_{AV}=\cdot555$ | ·058 | ·-0 | | | ·060 | ·006 | | | ·061 | ·0020 | | | ·0008 |
| | | | | | $r_{UO}=\cdot-0$ | | | | $r_{AO.L}=\cdot770$ | | | | $r_{AU.L}=\cdot220$ | | | | $r_{AV.L}=-\cdot132$ | |
| Original Standard Deviation of Mean of L | ·160 | ·160 | ·160 | ·160 | $r_{UV}=\cdot439$ | | | ·160 | | | | ·160 | | | | ·160 | | |
| Standard Deviation from Regression | ·104 | ·104 | ·119 | ·100 | | | | ·104 | | | | ·096 | | | | ·076 | | |
| "Explained" Variance | 59·3% | ·-0% | 46·1% | 62·1% | | 59·3% | ·-0% | 59·3% | | 43·8% | 21·4% | 65·2% | | 41·0% | 37·1% | 78·1% | | 63·5% |
| Variance Ratio F | 52·5 | ·-0 | 30·8 | 57·2 | | | | 52·5 | | | | 32·8 | | | | 62·4 | | |
| NOTIFICATION RATE: N | | | | | | | | | | | | | | | | | | |
| Total Correlation Coefficient | $r_{NA}=\cdot708$ | $r_{NO}=\cdot-0$ | $r_{NU}=\cdot644$ | $r_{NV}=\cdot568$ | | | | $R=\cdot708$ | | | | $R=\cdot750$ | | | | $R=\cdot743$ | | |
| Partial Correlation Coefficient $r$ | | | | | $r_{AO}=\cdot-0$ | ·708 | ·-0 | | | 591 | ·351 | | | ·619 | ·304 | | | ·608 |
| Regression Coefficient $b$ | $b_{NA}=10\cdot36$ | $b_{NO}=\cdot-0$ | $b_{NU}=\cdot779$ | $b_{NV}=\cdot238$ | $r_{AU}=\cdot640$ | 10·36 | ·-0 | | | 7·34 | ·391 | | | 8·91 | ·089 | | | ·133 |
| Standard Error of Regression Coefficient | 1·75 | ·-0 | ·156 | ·058 | $r_{AV}=\cdot555$ | 1·75 | ·-0 | | | 1·72 | ·176 | | | 1·94 | ·047 | | | ·029 |
| | | | | | $r_{UO}=\cdot-0$ | | | | $r_{AO.N}=\cdot708$ | | | | $r_{AU.N}=\cdot321$ | | | | $r_{AV.N}=\cdot028$ | |
| Original Standard Deviation of Mean of N | 4·343 | 4·343 | 4·343 | 4·343 | $r_{UV}=\cdot439$ | | | 4·343 | | | | 4·343 | | | | 4·343 | | |
| Standard Deviation from Regression | 3·111 | 3·111 | 3·371 | 3·624 | | | | 3·111 | | | | 2·913 | | | | 2·948 | | |
| "Explained" Variance | 50·1% | ·-0% | 41·4% | 32·3% | | 50·1% | ·-0% | 50·1% | | 35·5% | 20·8% | 56·3% | | 43·1% | 12·1% | 55·2% | | 42·1% |
| Variance Ratio F | 35·2 | ·-0 | 24·7 | 16·7 | | | | 35·2 | | | | 22·5 | | | | 21·6 | | |

## TABLE VII

1930–32: CORRELATIONS, REGRESSIONS, AND ANALYSES OF VARIANCE OF RESPIRATORY TUBERCULOSIS RATES
in relation to ORDINARY CROWDING, OVERCROWDING, UNEMPLOYMENT, and POVERTY in the 38 Wards of GLASGOW.

| th A and U | DOUBLE REGRESSION with A and V | | | | DOUBLE REGRESSION with T and U | | | | DOUBLE REGRESSION with T and V | | | | DOUBLE REGRESSION with U and V | | | | TRIPLE REGRESSION with A, T, U | | | | | TRIPLE REGRESS | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Inter-relations | Partial relation to | | Joint relation to A and V | Inter-relations | Partial relation to | | Joint relation to T and U | Inter-relations | Partial relation to | | Joint relation to T and V | Inter-relations | Partial relation to | | Joint relation to U and V | Inter-relations | Partial relation to | | | Joint relation to A, T, U | Inter-relations | Partial relation to | | | r |
| | A | V | | | T | U | | | T | V | | | U | V | | | A | T | U | | | A | T | V | |
| $r_{AU.M}=\cdot246$ | ·603<br>4·27<br>·955 | ·482<br>·116<br>·036 | $R=\cdot863$ | $r_{AV.M}=-\cdot167$ | ·781<br>·102<br>·014 | ·345<br>·161<br>·074 | $R=\cdot874$ | $r_{TU.M}=\cdot314$ | ·762<br>·100<br>·014 | ·356<br>·064<br>·028 | $R=\cdot883$ | $r_{TV.M}=\cdot347$ | ·551<br>·259<br>·066 | ·619<br>·142<br>·030 | $R=\cdot833$ | $r_{UV.M}=\cdot132$ | ·578<br>3·70<br>·896 | ·687<br>·077<br>·014 | ·145<br>·034<br>·040 | $R=\cdot909$ | $r_{AT.UM}=-\cdot015$<br>$r_{AU.TM}=\cdot216$<br>$r_{TU.AM}=\cdot329$ | ·584<br>3·65<br>·870 | ·625<br>·066<br>·014 | ·322<br>·040<br>·020 | R |
| | | | 2·457<br>1·261<br>74·4%<br>50·9 | | | | 2·457<br>1·211<br>76·4%<br>50·7 | | | | 2·457<br>1·174<br>77·9%<br>61·7 | | | | 2·457<br>1·378<br>69·4%<br>39·7 | | | | | 2·457<br>1·052<br>82·7%<br>54·2 | | | | | |
| | 39·1% | 35·3% | | | 60·1% | 16·3% | | | 58·4% | 19·5% | | | 26·2% | 43·2% | | | 33·8% | 45·5% | 3·4% | | | 33·4% | 38·7% | 12·2% | |
| $r_{AU.L}=\cdot220$ | ·632<br>·287<br>·061 | ·531<br>·0073<br>·0020 | $R=\cdot884$ | $r_{AV.L}=-\cdot132$ | ·804<br>·0061<br>·0008 | ·412<br>·0096<br>·0036 | $R=\cdot885$ | $r_{TU.L}=\cdot281$ | ·798<br>·0059<br>·0008 | ·512<br>·0038<br>·0011 | $R=\cdot894$ | $r_{TV.L}=\cdot349$ | ·602<br>·0184<br>·0041 | ·722<br>·0094<br>·0015 | $R=\cdot870$ | $r_{UV.L}=-\cdot211$ | ·617<br>·254<br>·053 | ·738<br>·0046<br>·0007 | ·073<br>·0018<br>·0042 | $R=\cdot934$ | $r_{AT.UL}=-\cdot002$<br>$r_{AU.TL}=\cdot217$<br>$r_{TU.AL}=\cdot303$ | ·610<br>·250<br>·054 | ·664<br>·0036<br>·0007 | ·505<br>·0031<br>·0009 | R |
| | | | ·160<br>·076<br>78·1%<br>62·4 | | | | ·160<br>·076<br>78·3%<br>63·1 | | | | ·160<br>·073<br>79·9%<br>69·6 | | | | ·160<br>·080<br>75·7%<br>54·5 | | | | | ·160<br>·059<br>87·2%<br>77·2 | | | | | |
| | 41·0% | 37·1% | | | 63·5% | 14·8% | | | 61·1% | 18·8% | | | 28·0% | 47·7% | | | 36·3% | 48·1% | 2·8% | | | 35·7% | 37·2% | 15·9% | |
| $r_{AU.N}=\cdot321$ | ·619<br>8·91<br>1·94 | ·304<br>·089<br>·047 | $R=\cdot743$ | $r_{AV.N}=\cdot028$ | ·608<br>·133<br>·029 | ·334<br>·355<br>·169 | $R=\cdot781$ | $r_{TU.N}=\cdot267$ | ·628<br>·143<br>·030 | ·316<br>·107<br>·054 | $R=\cdot774$ | $r_{TV.N}=\cdot340$ | ·535<br>·528<br>·143 | ·427<br>·143<br>·051 | $R=\cdot689$ | $r_{UV.N}=-\cdot163$ | ·524<br>4·51<br>1·26 | ·571<br>·110<br>·027 | ·212<br>·263<br>·208 | $R=\cdot841$ | $r_{AT.UN}=-\cdot133$<br>$r_{AU.TN}=\cdot167$<br>$r_{TU.AN}=\cdot285$ | ·551<br>4·73<br>1·23 | ·590<br>·118<br>·028 | ·168<br>·062<br>·062 | R |
| | | | 4·343<br>2·948<br>55·2%<br>21·6 | | | | 4·343<br>2·751<br>61·0%<br>27·4 | | | | 4·343<br>2·789<br>59·9%<br>26·1 | | | | 4·343<br>3·193<br>47·5%<br>15·8 | | | | | 4·343<br>2·418<br>70·7%<br>27·4 | | | | | |
| | 43·1% | 12·1% | | | 42·1% | 18·9% | | | 45·4% | 14·5% | | | 28·1% | 19·4% | | | 21·8% | 34·9% | 14·0% | | | 22·9% | 37·3% | 8·4% | |

## TABLE VIII

1936–38: CORRELATIONS, REGRESSIONS, AND ANALYSES OF VARIANCE OF RESPIRATORY TUBERCULOSIS RATES
in relation to ORDINARY CROWDING, OVERCROWDING, UNEMPLOYMENT, and POVERTY in the 38 Wards of GLASGOW.

| th A and U | DOUBLE REGRESSION with A and V | | | | DOUBLE REGRESSION with O and U | | | | DOUBLE REGRESSION with O and V | | | | DOUBLE REGRESSION with U and V | | | | TRIPLE REGRESSION with A, O, U | | | | | TRIPLE REGRESS | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Inter-relations | Partial relation to | | Joint relation to A and V | Inter-relations | Partial relation to | | Joint relation to O and U | Inter-relations | Partial relation to | | Joint relation to O and V | Inter-relations | Partial relation to | | Joint relation to U and V | Inter-relations | Partial relation to | | | Joint relation to A, O, U | Inter-relations | Partial relation to | | |
| | A | V | | | O | U | | | O | V | | | U | V | | | A | O | U | | | A | O | V |
| | | | | | | | | | | | | | | | | | | | | | | | | |
| | | | | | | | | | | | | | | | | | | | | | | | | |
| | | | | | | | | | | | | | | | | | | | | | | | |

RATES

| RIPLE REGRESSION with A, T, U | | | | TRIPLE REGRESSION with A, T, V | | | | | TRIPLE REGRESSION with A, U, V | | | | | TRIPLE REGRESSION with T, U, V | | | | | MULTIPLE RELATIONSHIP $M_e = 3{\cdot}586\,A + {\cdot}060\,T + {\cdot}012\,U + {\cdot}050\,V - 8{\cdot}978$ | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| elation to | | Joint relation to A, T, U | Inter-relations | Partial relation to | | | Joint relation to A, T, V | Inter-relations | Partial relation to | | | Joint relation to A, U, V | Inter-relations | Partial relation to | | | Joint relation to T, U, V | Inter-relations | Partial relation to | | | | Joint relation to A, T, U, V | Inter-relations |
| T | U | | | A | T | V | | | A | U | V | | | T | U | V | | | A | T | U | V | | |
| 687<br>077<br>014 | ·145<br>·034<br>·040 | $R = {\cdot}909$ | $r_{AT.UM} = -{\cdot}015$<br>$r_{AU.TM} = {\cdot}216$<br>$r_{TU.AM} = {\cdot}329$ | ·584<br>3·65<br>·870 | ·625<br>·066<br>·014 | ·322<br>·040<br>·020 | $R = {\cdot}918$ | $r_{AT.VM} = -{\cdot}021$<br>$r_{AV.TM} = -{\cdot}125$<br>$r_{TV.AM} = {\cdot}314$ | ·596<br>4·25<br>·982 | ·314<br>·150<br>·078 | ·445<br>·085<br>·029 | $R = {\cdot}894$ | $r_{AU.VM} = {\cdot}205$<br>$r_{AV.UM} = -{\cdot}118$<br>$r_{UV.AM} = -{\cdot}093$ | ·733<br>·095<br>·015 | ·135<br>·064<br>·081 | ·427<br>·064<br>·023 | $R = {\cdot}903$ | $r_{TU.VM} = {\cdot}242$<br>$r_{TV.UM} = {\cdot}291$<br>$r_{UV.TM} = -{\cdot}109$ | ·590<br>3·59<br>·830 | ·631<br>·060<br>·013 | ·085<br>·102<br>·024 | ·411<br>·050<br>·024 | $R = {\cdot}919$ | $r_{AT.UVM} = {\cdot}057$<br>$r_{AU.TVM} = {\cdot}174$<br>$r_{AV.TUM} = -{\cdot}101$<br>$r_{TU.AVM} = {\cdot}217$<br>$r_{TV.AUM} = {\cdot}278$<br>$r_{UV.ATM} = {\cdot}067$ |
| 5% | 3·4% | 2·457<br>1·052<br>82·7%<br>54·2 | | 33·4% | 38·7% | 12·2% | 2·457<br>1·002<br>84·3%<br>60·9 | | 38·9% | 15·1% | 25·9% | 2·457<br>1·134<br>79·9%<br>45·1 | | 55·7% | 6·5% | 19·4% | 2·457<br>1·085<br>81·6%<br>50·3 | | 32·8% | 35·4% | 1·2% | 15·1% | 2·457<br>1·010<br>84·5%<br>45·0 | |
| 738<br>0046<br>0007 | ·073<br>·0018<br>·0042 | $R = {\cdot}934$ | $r_{AT.UL} = -{\cdot}002$<br>$r_{AU.TL} = {\cdot}217$<br>$r_{TU.AL} = {\cdot}303$ | ·610<br>·250<br>·054 | ·664<br>·0036<br>·0007 | ·505<br>·0031<br>·0009 | $R = {\cdot}942$ | $r_{AT.VL} = -{\cdot}007$<br>$r_{AV.TL} = -{\cdot}091$<br>$r_{TV.AL} = {\cdot}316$ | ·645<br>·271<br>·055 | ·463<br>·011<br>·004 | ·543<br>·0052<br>·0014 | $R = {\cdot}903$ | $r_{AU.VL} = {\cdot}197$<br>$r_{AV.UL} = -{\cdot}067$<br>$r_{UV.AL} = -{\cdot}061$ | ·785<br>·0058<br>·0008 | ·169<br>·0036<br>·0037 | ·516<br>·0042<br>·0011 | $R = {\cdot}932$ | $r_{TU.VL} = {\cdot}222$<br>$r_{TV.UL} = {\cdot}302$<br>$r_{UV.TL} = -{\cdot}143$ | ·609<br>·248<br>·053 | ·644<br>·0034<br>·0007 | ·064<br>·0013<br>·0033 | ·505<br>·0032<br>·0009 | $R = {\cdot}943$ | $r_{AT.UVL} = -{\cdot}002$<br>$r_{AU.TVL} = {\cdot}205$<br>$r_{AV.TUL} = -{\cdot}093$<br>$r_{TU.AVL} = {\cdot}206$<br>$r_{TV.AUL} = {\cdot}269$<br>$r_{UV.ATL} = -{\cdot}093$ |
| 1% | 2·8% | ·160<br>·059<br>87·2%<br>77·2 | | 35·7% | 37·2% | 15·9% | ·160<br>·055<br>88·8%<br>89·9 | | 38·7% | 16·6% | 26·3% | ·160<br>·071<br>81·6%<br>50·3 | | 60·3% | 5·3% | 21·3% | ·160<br>·059<br>86·9%<br>75·2 | | 35·3% | 35·1% | 2·0% | 16·5% | ·160<br>·056<br>88·9%<br>66·8 | |
| 571<br>110<br>027 | ·212<br>·263<br>·208 | $R = {\cdot}841$ | $r_{AT.UN} = -{\cdot}133$<br>$r_{AU.TN} = {\cdot}167$<br>$r_{TU.AN} = {\cdot}285$ | ·551<br>4·73<br>1·23 | ·590<br>·118<br>·028 | ·168<br>·062<br>·062 | $R = {\cdot}834$ | $r_{AT.VN} = {\cdot}048$<br>$r_{AV.TN} = -{\cdot}004$<br>$r_{TV.AN} = {\cdot}327$ | ·602<br>7·09<br>1·61 | ·289<br>·357<br>·203 | ·191<br>·073<br>·064 | $R = {\cdot}795$ | $r_{AU.VN} = {\cdot}181$<br>$r_{AV.UN} = {\cdot}051$<br>$r_{UV.AN} = -{\cdot}040$ | ·613<br>·128<br>·028 | ·266<br>·338<br>·210 | ·183<br>·069<br>·064 | $R = {\cdot}824$ | $r_{TU.VN} = {\cdot}297$<br>$r_{TV.UN} = {\cdot}331$<br>$r_{UV.TN} = {\cdot}057$ | ·527<br>4·36<br>1·22 | ·570<br>·106<br>·027 | ·208<br>·222<br>·182 | ·112<br>·039<br>·060 | $R = {\cdot}847$ | $r_{AT.UVN} = {\cdot}031$<br>$r_{AU.TVN} = {\cdot}188$<br>$r_{AV.TUN} = -{\cdot}041$<br>$r_{TU.AVN} = {\cdot}215$<br>$r_{TV.AUN} = {\cdot}264$<br>$r_{UV.ATN} = -{\cdot}063$ |
| 9% | 14·0% | 4·343<br>2·418<br>70·7%<br>27·4 | | 22·9% | 37·3% | 8·4% | 4·343<br>2·466<br>69·6%<br>25·9 | | 34·3% | 19·0% | 9·9% | 4·343<br>2·711<br>63·2%<br>19·5 | | 40·5% | 18·0% | 9·4% | 4·343<br>2·532<br>67·9%<br>24·0 | | 21·1% | 33·5% | 11·8% | 5·3% | 4·343<br>2·411<br>71·7%<br>20·9 | |

RATES

| RIPLE REGRESSION with A, O, U | | | | TRIPLE REGRESSION with A, O, V | | | | | TRIPLE REGRESSION with A, U, V | | | | | TRIPLE REGRESSION with O, U, V | | | | | MULTIPLE REGRESSION | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| elation to | | Joint relation to A, O, U | Inter-relations | Partial relation to | | | Joint relation to A, O, V | Inter-relations | Partial relation to | | | Joint relation to A, U, V | Inter-relations | Partial relation to | | | Joint relation to O, U, V | Inter-relations | Partial relation to | | | | Joint relation to A, O, U, V | Inter-relations |
| O | U | | | A | O | V | | | A | U | V | | | O | U | V | | | A | O | U | V | | |

| | | | | | | | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| LOG. OF MORTALITY RATE: $^{L}M$ | | | | | | | | | | | | | | | | | | | | | | | | |
| Total Correlation Coefficient | $r_{LA}=\cdot770$ | $r_{LT}=\cdot823$ | $r_{LU}=\cdot679$ | $r_{LV}=\cdot788$ | $r_{AT}=\cdot731$ | ·618 | ·744 | $R=\cdot933$ | | ·656 | ·381 | $R=\cdot807$ | | ·632 | ·531 | $R=\cdot884$ | | ·804 | ·412 | $R=\cdot885$ | | | | |
| Partial Correlation Coefficient $r$ | | | | | $r_{AU}=\cdot640$ | ·257 | ·0049 | | | ·307 | ·014 | | | ·287 | ·0073 | | | ·0061 | ·0096 | | | | | |
| Regression Coefficient $b$ | $b_{LA}=\cdot416$ | $b_{LT}=\cdot0065$ | $b_{LV}=\cdot0303$ | $b_{LV}=\cdot0122$ | $r_{AV}=\cdot555$ | ·054 | ·0007 | | $r_{AT.L}=\cdot081$ | ·060 | ·006 | | $r_{AU.L}=\cdot220$ | ·061 | ·0020 | | $r_{AV.L}=-\cdot132$ | ·0008 | ·0036 | | $r_{TU.L}=\cdot28$ | | | |
| Standard Error of Regression Coefficient | ·058 | ·0007 | ·0055 | ·0016 | $r_{TU}=\cdot702$ | | | | | | | | | | | | | | | | | | | |
| Original Standard Deviation of Mean of L | ·160 | ·160 | ·160 | ·160 | $r_{TV}=\cdot763$ | | | | | | | | | | | | | | | | | | | |
| Standard Deviation from Regression | ·104 | ·091 | ·119 | ·100 | $r_{UV}=\cdot439$ | | | ·160 | | | | ·160 | | | | ·160 | | | | ·160 | | | | |
| "Explained" Variance | 59·3% | 67·8% | 46·1% | 62·1% | | 36·3% | 50·8% | ·058 | | 43·8% | 21·4% | ·096 | | 41·0% | 37·1% | ·076 | | 63·5% | 14·8% | ·076 | | | | |
| Variance Ratio F | 52·5 | 75·8 | 30·8 | 57·2 | | | | 87·1% | | | | 65·2% | | | | 78·1% | | | | 78·3% | | | | |
| | | | | | | | | 118·2 | | | | 32·8 | | | | 62·4 | | | | 63·1 | | | | |
| NOTIFICATION RATE: N | | | | | | | | | | | | | | | | | | | | | | | | |
| Total Correlation Coefficient | $r_{NA}=\cdot708$ | $r_{NT}=\cdot760$ | $r_{NU}=\cdot644$ | $r_{NV}=\cdot568$ | $r_{AT}=\cdot731$ | ·540 | ·583 | $R=\cdot792$ | | 591 | ·351 | $R=\cdot750$ | | ·619 | ·304 | $R=\cdot743$ | | ·608 | ·334 | $R=\cdot781$ | | | | |
| Partial Correlation Coefficient $r$ | | | | | $r_{AU}=\cdot640$ | 4·80 | ·125 | | | 7·34 | ·391 | | | 8·91 | ·089 | | | ·133 | ·355 | | | | | |
| Regression Coefficient $b$ | $b_{NA}=10\cdot36$ | $b_{NT}=\cdot182$ | $b_{NU}=\cdot779$ | $b_{NV}=\cdot238$ | $r_{AV}=\cdot555$ | 1·27 | ·029 | | $r_{AT.N}=\cdot320$ | 1·72 | ·176 | | $r_{AU.N}=\cdot321$ | 1·94 | ·047 | | $r_{AV.N}=\cdot028$ | ·029 | ·169 | | $r_{TU.N}=$ | | | |
| Standard Error of Regression Coefficient | 1·75 | ·026 | ·156 | ·058 | $r_{TU}=\cdot702$ | | | | | | | | | | | | | | | | | | | |
| Original Standard Deviation of Mean of N | 4·343 | 4·343 | 4·343 | 4·343 | $r_{TV}=\cdot763$ | | | ·792 | | | | ·750 | | | | ·743 | | | | ·781 | | | | |
| Standard Deviation from Regression | 3·111 | 2·864 | 3·371 | 3·624 | $r_{UV}=\cdot439$ | | | | | | | | | | | | | | | | | | | |
| "Explained" Variance | 50·1% | 57·7% | 41·4% | 32·3% | | 23·2% | 39·5% | 4·343 | | 35·5% | 20·8% | 4·343 | | 43·1% | 12·1% | 4·343 | | 42·1% | 18·9% | 4·343 | | | | |
| Variance Ratio F | 35·2 | 47·8 | 24·7 | 16·7 | | | | 2·728 | | | | 2·913 | | | | 2·948 | | | | 2·751 | | | | |
| | | | | | | | | 62·7% | | | | 56·3% | | | | 55·2% | | | | 61·0% | | | | |
| | | | | | | | | 28·6 | | | | 22·5 | | | | 21·6 | | | | 27·4 | | | | |



| | SINGLE REGRESSIONS | | | | | DOUBLE REGRESSION with A and O | | | | DOUBLE REGRESSION with A and U | | | | DOUBLE REGRESSION with A and V | | | | DOUBLE REGRESSION with O and | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Relation to | | | | Inter-relations of Social Factors | Partial relation to | | Joint relation to A and O | Inter-relations | Partial relation to | | Joint relation to A and U | Inter-relations | Partial relation to | | Joint relation to A and V | Inter-relations | Partial relation to | | Joint relation to O and U | Inter-r |
| | A | O | U | V | | A | O | | | A | U | | | A | V | | | O | U | | |
| MORTALITY RATE: M | | | | | | | | | | | | | | | | | | | | | |
| Total Correlation Coefficient | $r_{MA}=\cdot809$ | $r_{MO}=\cdot772$ | $r_{MU}=\cdot622$ | $r_{MV}=\cdot640$ | $r_{AO}=\cdot578$ | ·699 | ·634 | $R=\cdot891$ | | ·703 | ·321 | $R=\cdot831$ | | ·713 | ·401 | $R=\cdot842$ | | ·594 | ·138 | $R=\cdot777$ | |
| Partial Correlation Coefficient $r$ | | | | | $r_{AU}=\cdot579$ | 4·85 | ·086 | | | 6·01 | ·212 | | | 5·83 | ·072 | | | ·127 | ·118 | | |
| Regression Coefficient $b$ | $b_{MA}=7\cdot201$ | $b_{MO}=\cdot144$ | $b_{MU}=\cdot571$ | $b_{MV}=\cdot163$ | $r_{AV}=\cdot547$ | ·838 | ·018 | | | 1·03 | ·106 | | | ·970 | ·028 | | | ·029 | ·143 | | |
| Standard Error of Regression Coefficient | ·872 | ·020 | ·120 | ·033 | $r_{OU}=\cdot728$ | | | | $r_{AO.M}=-\cdot124$ | | | | $r_{AU.M}=\cdot164$ | | | | $r_{AV.M}=-\cdot065$ | | | | $r_{O}$ |
| Original Standard Deviation of Mean of M | 2·622 | 2·622 | 2·622 | 2·622 | $r_{OV}=\cdot709$ | | | 2·622 | | | | 2·622 | | | | 2·622 | | | | 2·622 | |
| Standard Deviation from Regression | 1·563 | 1·690 | 2·081 | 2·043 | $r_{UV}=\cdot435$ | | | 1·225 | | | | 1·501 | | | | 1·452 | | | | 1·698 | |
| "Explained" Variance | 65·4% | 59·6% | 38·7% | 40·9% | | 44·1% | 35·2% | 79·3% | | 54·6% | 14·4% | 69·0% | | 53·0% | 18·0% | 71·0% | | 52·4% | 8·0% | 60·4% | |
| Variance Ratio F | 68·1 | 53·0 | 22·7 | 24·9 | | | | 67·2 | | | | 38·9 | | | | 42·8 | | | | 26·6 | |
| LOG. OF MORTALITY RATE: $^{L}M$ | | | | | | | | | | | | | | | | | | | | | |
| Total Correlation Coefficient | $r_{LA}=\cdot840$ | $r_{LO}=\cdot788$ | $r_{LU}=\cdot644$ | $r_{LV}=\cdot703$ | $r_{AO}=\cdot578$ | ·765 | ·683 | $R=\cdot918$ | | ·749 | ·356 | $R=\cdot862$ | | ·765 | ·535 | $R=\cdot889$ | | ·608 | ·167 | $R=\cdot795$ | |
| Partial Correlation Coefficient $r$ | | | | | $r_{AU}=\cdot579$ | ·335 | ·0054 | | | ·395 | ·014 | | | ·366 | ·0056 | | | ·0080 | ·0087 | | |
| Regression Coefficient $b$ | $b_{LA}=\cdot473$ | $b_{LO}=\cdot0093$ | $b_{LU}=\cdot037$ | $b_{LV}=\cdot011$ | $r_{AV}=\cdot547$ | ·046 | ·0010 | | | ·059 | ·006 | | | ·052 | ·0015 | | | ·0018 | ·0087 | | |
| Standard Error of Regression Coefficient | ·051 | ·0012 | ·007 | ·002 | $r_{OU}=\cdot728$ | | | | $r_{AO.L}=-\cdot251$ | | | | $r_{AU.L}=\cdot092$ | | | | $r_{AV.L}=-\cdot111$ | | | | |
| Original Standard Deviation of Mean of L | ·166 | ·166 | ·166 | ·166 | $r_{OV}=\cdot709$ | | | ·166 | | | | ·166 | | | | ·166 | | | | ·166 | |
| Standard Deviation from Regression | ·091 | ·104 | ·129 | ·120 | $r_{UV}=\cdot435$ | | | ·068 | | | | ·096 | | | | ·078 | | | | ·104 | |
| "Explained" Variance | 70·5% | 62·0% | 41·5% | 49·4% | | 48·5% | 35·8% | 84·3% | | 59·0% | 15·3% | 74·3% | | 54·6% | 24·4% | 79·0% | | 53·5% | 9·6% | 63·1% | |
| Variance Ratio F | 86·2 | 58·9 | 25·5 | 35·1 | | | | 93·8 | | | | 50·5 | | | | 65·7 | | | | 30·0 | |
| NOTIFICATION RATE: N | | | | | | | | | | | | | | | | | | | | | |
| Total Correlation Coefficient | $r_{NA}=\cdot757$ | $r_{NO}=\cdot823$ | $r_{NU}=\cdot734$ | $r_{NV}=\cdot596$ | $r_{AO}=\cdot578$ | ·607 | ·724 | $R=\cdot893$ | | ·600 | ·554 | $R=\cdot839$ | | ·641 | ·333 | $R=\cdot788$ | | ·621 | ·345 | $R=\cdot846$ | |
| Partial Correlation Coefficient $r$ | | | | | $r_{AU}=\cdot579$ | 6·36 | ·184 | | | 7·53 | ·691 | | | 9·27 | ·112 | | | ·195 | ·444 | | |
| Regression Coefficient $b$ | $b_{NA}=11\cdot41$ | $b_{NO}=\cdot261$ | $b_{NU}=1\cdot14$ | $b_{NV}=\cdot257$ | $r_{AV}=\cdot547$ | 1·41 | ·030 | | | 1·70 | ·175 | | | 1·88 | ·054 | | | ·042 | ·204 | | |
| Standard Error of Regression Coefficient | 1·64 | ·030 | ·176 | ·058 | $r_{OU}=\cdot728$ | | | | $r_{AO.N}=-\cdot122$ | | | | $r_{AU.N}=\cdot053$ | | | | $r_{AV.N}=\cdot182$ | | | | |
| Original Standard Deviation of Mean of N | 4·44 | 4·44 | 4·44 | 4·44 | $r_{OV}=\cdot709$ | | | 4·44 | | | | 4·44 | | | | 4·44 | | | | 4·44 | |
| Standard Deviation from Regression | 2·94 | 2·56 | 3·06 | 3·61 | $r_{UV}=\cdot435$ | | | 2·06 | | | | 2·48 | | | | 2·81 | | | | 2·43 | |
| "Explained" Variance | 57·3% | 67·8% | 53·8% | 35·6% | | 32·0% | 47·7% | 79·72% | | 37·8% | 32·6% | 70·4% | | 45·5% | 15·5% | 62·0% | | 50·6% | 21·0% | 71·6% | |
| Variance Ratio F | 48·3 | 75·7 | 42·0 | 19·9 | | | | 68·5 | | | | 41·7 | | | | 28·6 | | | | 44·2 | |

| | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
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| | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| | | ·798 | ·512 | $R = \cdot 894$ | | ·602 | ·722 | $R = \cdot 870$ | | ·617 | ·738 | ·073 | $R = \cdot 934$ | | ·610 | ·664 | ·505 | $R = \cdot 942$ | | ·645 | ·463 | ·543 | $R = \cdot 903$ | | | | | | | |
| | $r_{TU.L} = \cdot 281$ | ·0059 | ·0038 | | $r_{TV.L} = \cdot 349$ | ·0184 | ·0094 | | $r_{UV.L} = -\cdot 211$ | 4·54 | ·0046 | ·0018 | | $r_{AT.UL} = -\cdot 002$ | ·250 | ·0036 | ·0031 | | $r_{AT.VL} = -\cdot 007$ | ·271 | ·011 | ·0052 | | $r_{AU.VL} =$ | | | | | |
| | | ·0008 | ·0011 | | | ·0041 | ·0015 | | | 1·26 | ·0007 | ·0042 | | $r_{AU.TL} = \cdot 217$ | ·054 | ·0007 | ·0009 | | $r_{AV.TL} = -\cdot 091$ | ·055 | ·004 | ·0014 | | $r_{AV.UL} =$ | | | | | |
| | | | | ·160 | | | | ·160 | | | | | ·160 | $r_{TU.AL} = \cdot 303$ | | | | ·160 | $r_{TV.AL} = \cdot 316$ | | | | ·160 | $r_{UV.AL} =$ | | | | | |
| | | 61·1% | 18·8% | ·073 | | 28·0% | 47·7% | ·080 | | 36·3% | 48·1% | 2·8% | ·059 | | 35·7% | 37·2% | 15·9% | ·055 | | 38·7% | 16·6% | 26·3% | ·071 | | | | | | |
| | | | | 79·9% | | | | 75·7% | | | | | 87·2% | | | | | 88·8% | | | | | 81·6% | | | | | | |
| | | | | 69·6 | | | | 54·5 | | | | | 77·2 | | | | | 89·9 | | | | | 50·3 | | | | | | |
| | | ·628 | ·316 | $R = \cdot 774$ | | ·535 | ·427 | $R = \cdot 689$ | | ·524 | ·571 | ·212 | $R = \cdot 841$ | | ·551 | ·590 | ·168 | $R = \cdot 834$ | | ·602 | ·289 | ·191 | $R = \cdot 795$ | | | | | | | |
| | $r_{TU.N} = \cdot 267$ | ·143 | ·107 | | $r_{TV.N} = \cdot 340$ | ·528 | ·143 | | $r_{UV.N} = -\cdot 163$ | 4·51 | ·110 | ·263 | | $r_{AT.UN} = -\cdot 133$ | 4·73 | ·118 | ·062 | | $r_{AT.VN} = \cdot 048$ | 7·09 | ·357 | ·203 | | $r_{AU.VN} =$ | | | | | |
| | | ·030 | ·054 | | | ·143 | ·051 | | | 1·26 | ·027 | ·208 | | $r_{AU.TN} = \cdot 167$ | 1·23 | ·028 | ·062 | | $r_{AV.TN} = -\cdot 004$ | 1·61 | ·203 | ·064 | | $r_{AV.UN} =$ | | | | | |
| | | | | 4·343 | | | | 4·343 | | | | | 4·343 | $r_{TU.AN} = \cdot 285$ | | | | 4·343 | $r_{TV.AN} = \cdot 327$ | | | | 4·343 | $r_{UV.AN} =$ | | | | | |
| | | 45·4% | 14·5% | 2·789 | | 28·1% | 19·4% | 3·193 | | 21·8% | 34·9% | 14·0% | 2·418 | | 22·9% | 37·3% | 8·4% | 2·466 | | 34·3% | 19·0% | 9·9% | 2·711 | | | | | | |
| | | | | 59·9% | | | | 47·5% | | | | | 70·7% | | | | | 69·6% | | | | | 63·2% | | | | | | |
| | | | | 26·1 | | | | 15·8 | | | | | 27·4 | | | | | 25·9 | | | | | 19·5 | | | | | | |

## TABLE VIII

CORRELATIONS, REGRESSIONS, AND ANALYSES OF VARIANCE OF RESPIRATORY TUBERCULOSIS RATES
relation to ORDINARY CROWDING, OVERCROWDING, UNEMPLOYMENT, and POVERTY in the 38 Wards of GLASGOW.

| with O and U | | DOUBLE REGRESSION with O and V | | | | DOUBLE REGRESSION with U and V | | | | TRIPLE REGRESSION with A, O, U | | | | | TRIPLE REGRESSION with A, O, V | | | | | TRIPLE REGRESSION with A, U, V | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Inter-relations | Partial relation to | | Joint relation to O and V | Inter-relations | Partial relation to | | Joint relation to U and V | Inter-relations | Partial relation to | | | Joint relation to A, O, U | Inter-relations | Partial relation to | | | Joint relation to A, O, V | Inter-relations | Partial relation to | | | Joint relation to A, U, V | Inter-re | |
| | | O | V | | | U | V | | | A | O | U | | | A | O | V | | | A | U | V | | | |
| | | ·587 | ·206 | $R = \cdot 783$ | | ·515 | ·604 | $R = \cdot 780$ | | ·696 | ·629 | ·059 | $R = \cdot 893$ | | ·684 | ·529 | ·166 | $R = \cdot 891$ | | ·702 | ·267 | ·382 | $R = \cdot 859$ | | | | | | |
| | $r_{OU.M} = \cdot 428$ | ·120 | ·047 | | $r_{OV.M} = \cdot 404$ | ·413 | ·130 | | $r_{UV.M} = +\cdot 034$ | 4·82 | ·084 | ·018 | | $r_{AO.UM} = -\cdot 137$ | 4·67 | ·073 | ·028 | | $r_{AO.VM} = -\cdot 171$ | 5·08 | ·156 | ·067 | | $r_{AU.VM} =$ | | | | | |
| | | ·028 | ·038 | | | ·116 | ·029 | | | ·846 | ·018 | ·052 | | $r_{AU.OM} = \cdot 252$ | ·854 | ·020 | ·029 | | $r_{AV.OM} = -\cdot 135$ | ·890 | ·097 | ·028 | | $r_{AV.UM} =$ | | | | | |
| | | | | 2·622 | | | | 2·622 | | | | | 2·622 | $r_{OU.AM} = \cdot 412$ | | | | 2·622 | $r_{OV.AM} = \cdot 413$ | | | | 2·622 | $r_{UV.AM} =$ | | | | | |
| | | 49·4% | 11·9% | 1·677 | | 28·0% | 32·8% | 1·687 | | 43·8% | 34·8% | 1·2% | 1·229 | | 42·4% | 30·6% | 6·4% | 1·241 | | 46·2% | 10·6% | 16·9% | 1·403 | | | | | | |
| | | | | 61·3% | | | | 60·8% | | | | | 79·8% | | | | | 79·4% | | | | | 73·7% | | | | | | |
| | | | | 27·7 | | | | 27·2 | | | | | 44·8 | | | | | 43·7 | | | | | 31·8 | | | | | | |
| | | ·577 | ·332 | $R = \cdot 814$ | | ·528 | ·613 | $R = \cdot 797$ | | ·759 | ·627 | ·081 | $R = \cdot 919$ | | ·749 | ·543 | ·238 | $R = \cdot 923$ | | ·694 | ·309 | ·514 | $R = \cdot 900$ | | | | | | |
| | $r_{OU.L} = \cdot 418$ | ·0069 | ·0047 | | $r_{OV.L} = \cdot 354$ | ·024 | ·0084 | | $r_{UV.L} = \cdot 032$ | ·332 | ·0057 | ·0028 | | $r_{AO.UL} = -\cdot 314$ | ·309 | ·0044 | ·0022 | | $r_{AO.VL} = -\cdot 228$ | ·316 | ·011 | ·0051 | | $r_{AU.V} =$ | | | | | |
| | | ·0016 | ·0022 | | | ·007 | ·0018 | | | ·049 | ·0012 | ·0060 | | $r_{AU.OL} = \cdot 215$ | ·047 | ·0012 | ·0015 | | $r_{AV.OL} = -\cdot 025$ | ·056 | ·006 | ·0015 | | $r_{AV.U} =$ | | | | | |
| | | | | ·166 | | | | ·166 | | | | | ·166 | $r_{OU.AL} = \cdot 409$ | | | | ·166 | $r_{OV.AL} = \cdot 319$ | | | | ·166 | $r_{UV.A} =$ | | | | | |
| | | 45·9% | 20·3% | ·099 | | 26·9% | 36·6% | ·103 | | 47·6% | 35·3% | 1·5% | ·068 | | 46·1% | 29·4% | 9·7% | ·067 | | 46·8% | 11·9% | 22·4% | ·075 | | | | | | |
| | | | | 66·2% | | | | 63·5% | | | | | 84·4% | | | | | 85·2% | | | | | 81·1% | | | | | | |
| | | | | 34·3 | | | | 30·4 | | | | | 61·3 | | | | | 65·1 | | | | | 48·6 | | | | | | |
| | | ·698 | ·101 | $R = \cdot 824$ | | ·639 | ·453 | $R = \cdot 796$ | | ·566 | ·590 | ·228 | $R = \cdot 898$ | | ·618 | ·631 | ·117 | $R = \cdot 895$ | | ·511 | ·535 | ·289 | $R = \cdot 854$ | | | | | | |
| | $r_{OU.N} = \cdot 321$ | ·240 | ·034 | | $r_{OV.N} = \cdot 418$ | ·909 | ·147 | | $r_{UV.N} = \cdot 005$ | 5·80 | ·154 | ·243 | | $r_{AO.UN} = -\cdot 145$ | 6·67 | ·163 | ·028 | | $r_{AO.VN} = -\cdot 212$ | 6·25 | ·638 | ·082 | | $r_{AU.} =$ | | | | | |
| | | ·043 | ·058 | | | ·185 | ·049 | | | 1·41 | ·036 | ·178 | | $r_{AU.ON} = \cdot 098$ | 1·46 | ·034 | ·041 | | $r_{AV.ON} = -\cdot 246$ | 1·80 | ·173 | ·047 | | $r_{AV.} =$ | | | | | |
| | | | | 4·44 | | | | 4·44 | | | | | 4·44 | $r_{OU.AN} = \cdot 330$ | | | | 4·44 | $r_{OV.AN} = \cdot 413$ | | | | 4·44 | $r_{UV.} =$ | | | | | |
| | | 62·6% | 5·2% | 2·59 | | 42·9% | 20·4% | 2·76 | | 29·1% | 40·1% | 11·4% | 2·03 | | 35·5% | 42·6% | 4·0% | 2·07 | | 31·4% | 30·1% | 11·4% | 2·41 | | | | | | |
| | | | | 67·8% | | | | 63·3% | | | | | 80·7% | | | | | 80·1% | | | | | 72·9% | | | | | | |
| | | | | 36·9 | | | | 30·2 | | | | | 47·4 | | | | | 45·6 | | | | | 30·5 | | | | | | |

| -relations | TRIPLE REGRESSION with A, U, V | | | | | TRIPLE REGRESSION with O, U, V | | | | | MULTIPLE REGRESSION | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Partial relation to | | | Joint relation to A, U, V | Inter-relations | Partial relation to | | | Joint relation to O, U, V | Inter-relations | Partial relation to | | | | Joint relation to A, O, U, V | Inter-relations |
| | A | U | V | | | O | U | V | | | A | O | U | V | | |
| | | | | $R = \cdot 859$ | | | | | $R = \cdot 784$ | | | | | | $R = \cdot 895$ | |
| | ·702 | ·267 | ·382 | | | ·580 | ·125 | ·227 | | | ·689 | ·505 | ·069 | ·169 | | $r_{AO.UVM} = -\cdot 169$ |
| | 5·08 | ·156 | ·067 | | $r_{AU.VM} = \cdot 103$ | ·107 | ·100 | ·042 | | | 4·66 | ·072 | ·026 | ·025 | | $r_{AU.OVM} = \cdot 099$ |
| $_{VM} = -\cdot 171$ | ·890 | ·097 | ·028 | | $r_{AV.UM} = -\cdot 076$ | ·026 | ·136 | ·031 | | $r_{OU.VM} = \cdot 398$ | ·841 | ·021 | ·064 | ·025 | | $r_{AV.OUM} = -\cdot 063$ |
| $_{OM} = -\cdot 135$ | | | | | $r_{UV.AM} = -\cdot 025$ | | | | | $r_{OV.UM} = \cdot 345$ | | | | | | $r_{OU.AVM} = \cdot 384$ |
| $_{AM} = \cdot 413$ | | | | | | | | | | $r_{UV.OM} = -\cdot 110$ | | | | | | $r_{OV.AUM} = \cdot 338$ |
| | | | | 2·622 | | | | | 2·622 | | | | | | 2·622 | $r_{UV.AOM} = -\cdot 022$ |
| | 46·2% | 10·6% | 16·9% | 1·403 | | 44·0% | 6·8% | 10·6% | 1·699 | | 42·3% | 29·7% | 1·8% | 6·3% | 1·239 | |
| | | | | 73·7% | | | | | 61·4% | | | | | | 80·1% | |
| | | | | 31·8 | | | | | 18·0 | | | | | | 33·2 | |
| | | | | $R = \cdot 900$ | | | | | $R = \cdot 826$ | | | | | | $R = \cdot 923$ | |
| | ·694 | ·309 | ·514 | | | ·581 | ·221 | ·337 | | | ·731 | ·466 | ·022 | ·226 | | $r_{AO.UVL} = -\cdot 197$ |
| | ·316 | ·011 | ·0051 | | $r_{AU.VL} = \cdot 089$ | ·0064 | ·0072 | ·0040 | | | ·309 | ·0044 | ·0008 | ·0021 | | $r_{AU.OVL} = \cdot 073$ |
| $_{VL} = -\cdot 228$ | ·056 | ·006 | ·0015 | | $r_{AV.UL} = -\cdot 109$ | ·0016 | ·0056 | ·0019 | | $r_{OU.VL} = \cdot 423$ | ·051 | ·0015 | ·0061 | ·0016 | | $r_{AV.OUL} = -\cdot 023$ |
| $_{OL} = -\cdot 025$ | | | | | $r_{UV.AL} = -\cdot 022$ | | | | | $r_{OV.UL} = -\cdot 329$ | | | | | | $r_{OU.AVL} = \cdot 393$ |
| $_{AL} = \cdot 319$ | | | | | | | | | | $r_{UV.OL} = -\cdot 214$ | | | | | | $r_{OV.AUL} = \cdot 301$ |
| | | | | ·166 | | | | | ·166 | | | | | | ·166 | $r_{UV.AOL} = -\cdot 020$ |
| | 46·8% | 11·9% | 22·4% | ·075 | | 42·9% | 7·6% | 17·7% | ·098 | | 45·9% | 29·2% | 0·9% | 9·2% | ·067 | |
| | | | | 81·1% | | | | | 68·2% | | | | | | 85·2% | |
| | | | | 48·6 | | | | | 24·3 | | | | | | 47·4 | |
| | | | | $R = \cdot 854$ | | | | | $R = \cdot 848$ | | | | | | $R = \cdot 908$ | |
| | ·511 | ·535 | ·289 | | | ·481 | ·356 | ·122 | | | ·571 | ·588 | ·212 | ·112 | | $r_{AO.UVN} = -\cdot 118$ |
| | 6·25 | ·638 | ·082 | | $r_{AU.VN} = \cdot 042$ | ·171 | ·464 | ·039 | | $r_{OU.VN} = \cdot 329$ | 5·91 | ·151 | ·217 | ·022 | | $r_{AU.OVN} = \cdot 033$ |
| $_{VN} = -\cdot 212$ | 1·80 | ·173 | ·047 | | $r_{AV.UN} = -\cdot 163$ | ·053 | ·209 | ·054 | | $r_{OV.UN} = \cdot 431$ | 1·46 | ·035 | ·172 | ·033 | | $r_{AV.OUN} = -\cdot 136$ |
| $_{ON} = -\cdot 246$ | | | | | $r_{UV.AN} = \cdot 002$ | | | | | $r_{UV.ON} = \cdot 187$ | | | | | | $r_{OU.AVN} = \cdot 318$ |
| $_{AN} = \cdot 413$ | | | | 4·44 | | | | | 4·44 | | | | | | 4·44 | $r_{OV.AUN} = \cdot 401$ |
| | 31·4% | 30·1% | 11·4% | 2·41 | | 44·4% | 21·9% | 5·6% | 2·46 | | 29·6% | 39·3% | 10·2% | 3·3% | 1·97 | $r_{UV.AON} = \cdot 001$ |
| | | | | 72·9% | | | | | 71·9% | | | | | | 82·4% | |
| | | | | 30·5 | | | | | 29·0 | | | | | | 38·8 | |

| | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | | | | | $L_e = \cdot 248\,A + \cdot 0034\,T + \cdot 0013\,U + \cdot 0032\,V - \cdot 272$ | | | | | |
| | | | | $R = \cdot 903$ | | | | | $R = \cdot 932$ | | | | | | $R = \cdot 943$ | |
| | ·645 | ·463 | ·543 | | | ·785 | ·169 | ·516 | | | ·609 | ·644 | ·064 | ·505 | | $r_{AT.UVL} = -\cdot 002$ |
| | ·271 | ·011 | ·0052 | | $r_{AU.VL} = \cdot 197$ | ·0058 | ·0036 | ·0042 | | | ·248 | ·0034 | ·0013 | ·0032 | | $r_{AU.TVL} = \cdot 205$ |
| $_{VL} = -\cdot 007$ | ·055 | ·004 | ·0014 | | $r_{AV.UL} = -\cdot 067$ | ·0008 | ·0037 | ·0011 | | $r_{TU.VL} = \cdot 222$ | ·053 | ·0007 | ·0033 | ·0009 | | $r_{AV.TUL} = -\cdot 093$ |
| $_{TL} = -\cdot 091$ | | | | | $r_{UV.AL} = -\cdot 061$ | | | | | $r_{TV.UL} = \cdot 302$ | | | | | | $r_{TU.AVL} = \cdot 206$ |
| $_{AL} = \cdot 316$ | | | | | | | | | | $r_{UV.TL} = -\cdot 143$ | | | | | | $r_{TV.AUL} = \cdot 269$ |
| | | | | ·160 | | | | | ·160 | | | | | | ·160 | $r_{UV.ATL} = -\cdot 093$ |
| | 38·7% | 16·6% | 26·3% | ·071 | | 60·3% | 5·3% | 21·3% | ·059 | | 35·3% | 35·1% | 2·0% | 16·5% | ·056 | |
| | | | | 81·6% | | | | | 86·9% | | | | | | 88·9% | |
| | | | | 50·3 | | | | | 75·2 | | | | | | 66·8 | |
| | | | | | | | | | | | $N_e = 4\cdot 36\,A + \cdot 106\,T + \cdot 222\,U + \cdot 039\,V - 8\cdot 965$ | | | | | |
| | | | | $R = \cdot 795$ | | | | | $R = \cdot 824$ | | | | | | $R = \cdot 847$ | |
| | ·602 | ·289 | ·191 | | | ·613 | ·266 | ·183 | | | ·527 | ·570 | ·208 | ·112 | | $r_{AT.UVN} = \cdot 031$ |
| | 7·09 | ·357 | ·073 | | $r_{AU.VN} = \cdot 181$ | ·128 | ·338 | ·069 | | $r_{TU.VN} = \cdot 297$ | 4·36 | ·106 | ·222 | ·039 | | $r_{AU.TVN} = \cdot 188$ |
| $_{VN} = \cdot 048$ | 1·61 | ·203 | ·064 | | $r_{AV.UN} = \cdot 051$ | ·028 | ·210 | ·064 | | $r_{TV.UN} = \cdot 331$ | 1·22 | ·027 | ·182 | ·060 | | $r_{AV.TUN} = -\cdot 041$ |
| $_{TN} = -\cdot 004$ | | | | | $r_{UV.AN} = -\cdot 040$ | | | | | $r_{UV.TN} = \cdot 057$ | | | | | | $r_{TU.AVN} = \cdot 215$ |
| $_{AN} = \cdot 327$ | | | | 4·343 | | | | | 4·343 | | | | | | 4·343 | $r_{TV.AUN} = \cdot 264$ |
| | 34·3% | 19·0% | 9·9% | 2·711 | | 40·5% | 18·0% | 9·4% | 2·532 | | 21·1% | 33·5% | 11·8% | 5·3% | 2·411 | $r_{UV.ATN} = -\cdot 063$ |
| | | | | 63·2% | | | | | 67·9% | | | | | | 71·7% | |
| | | | | 19·5 | | | | | 24·0 | | | | | | 20·9 | |

post-war regression equations and in the diagrams which illustrate them. The inter-correlations between the social factors in the multiple equations indicate that there was almost no "overlap" between the distribution of ordinary crowding and the distributions of the other social variables. But some correspondence was found between poverty and over-crowding, and again between unemployment and overcrowding in the later 1930s. To some extent, the poverty and unemployment distributions were described by the overcrowding distribution: at the same time, there seemed to be sufficient dissimilarity between these three to make their contributions to regression different and of some size. It was surprising to find little correlation between average crowding and overcrowding, and only small residual correlations between ordinary crowding and poverty or unemployment. In most of the multiple equations, the partial correlations between ordinary crowding and overcrowding were very small and negative, and not significant. The inter-correlations between the other three social variables were of a higher order, but varied with the years. The correspondence between poverty and unemployment in the pre-war years was small, but increased in the post-war period.

PRE-WAR RELATIONSHIPS.—The estimated relationships of tuberculosis with the social distributions in various triennia in the 1930s were similar to those for 1930–32 (Table VII). They show, as do also the regressions for later periods, that the equations expressing the disease rates in terms of the social variables give a remarkably good "fit": standard errors of estimate (or standard deviations from regression) were small, and were always less than half the original standard deviations of means of mortality or notification rates. A still better "fit" was obtained in the regressions in which the logarithms of mortality rates were used, indicating again the upward curve of mortality as social conditions worsened. The equations for 1936–38 (Table VIII) not only indicate time-trends, but also include all four social variables. The estimated relationships of tuberculosis rates with these variables in 1936–38 were these:

$$M_e = 4{\cdot}655\,A + {\cdot}072\,O + {\cdot}026\,U + {\cdot}025\,V - 12{\cdot}791$$
$$L_e = {\cdot}309\,A + {\cdot}0044\,O + {\cdot}0008\,U + {\cdot}0021\,V - {\cdot}495$$
$$N_e = 5{\cdot}905\,A + {\cdot}151\,O + {\cdot}217\,U + {\cdot}022\,V - 14{\cdot}955$$

where $M_e$ represents estimated mortality rate (per 10,000 ward population).
$L_e$ represents estimated $\log_{10}$ of mortality rate.
$N_e$ represents estimated notification rate (per 10,000 population).
A represents ordinary crowding (ward average of persons per house).
O represents statutory overcrowding (percentage overcrowded houses).
U represents unemployment (adult unemployed men per cent. of population).
V represents poverty (valuation of ward according to rentals).

The high total correlations of mortality with the social combination and the large proportion of "explained" variance, particularly in the logarithmic regression, are shown in Table VIII. These relationships were remarkably similar to the post-war mortality relationships which will be illustrated later; the relative importance of the different social factors is apparent in the partial correlations of each factor with mortality or with notifications.

The notification relationship, represented by the third of the above equations, is illustrated in Fig. 5 (overleaf). The "explained" variance, 82 per cent. of the total, was again dominated by contributions from ordinary crowding and overcrowding of nearly 30 and 40 per cent. respectively, and the partial correlation with unemployment was also significant.

The diagram illustrates the contributions of each social factor, individually and in combination with other factors, to the variance "explained" in the notification relationship. The first four columns illustrate the direct correlations in individual association with notifications. In these four correlations could be perceived the final order of importance of the social variables; and this ranking re-appears in the

subsequent relationships when two or three social distributions are considered simultaneously. The second set of six columns, representing pairs of social factors, demonstrates also the extremely high proportion of variance "explained" by ordinary crowding coupled with overcrowding; other combinations gave a lesser correspondence with the distribution of notifications. The third set of six columns amplifies these conclusions, for it shows also that the earlier large contributions of overcrowding represented to some extent the contributions of poverty and unemployment. It is apparent that, though little was added to the "explained" variance when three social factors were combined, a clearer picture of the influence of each was presented.

The final column depicts the association between notification rates and the social complex in the wards. The contribution of poverty almost disappeared when the other social distributions were included, and the contribution of unemployment was still further reduced by the inclusion of poverty in the regression. It is manifest that, in this pre-war triennium, a single social variable did not adequately simulate the social complex in relation to tuberculosis. But a suitable pair of variables, ordinary crowding and overcrowding, gave in combination a good "model" which illustrated the total influence of the social complex on tuberculosis—a "model", however, which could not bring out the magnitude of the contributions of poverty and unemployment.

Post-War Relationships.—The preceding relationships for pre-war years gave some indications of the reduction in poverty and unemployment correlations when the overcrowding distribution was included in the equations. The post-war equations do not include overcrowding, since no data are available, and it is to be expected therefore that in the post-war regressions the contributions of poverty and unemployment to "explained" variance would be larger. The regressions for tuberculosis mortality and notification rates in terms of ward social conditions in the post-war years, however, are of interest at the present time. The summary in Table IX shows the high correlations and small standard deviations from regression, to which reference has already been made.

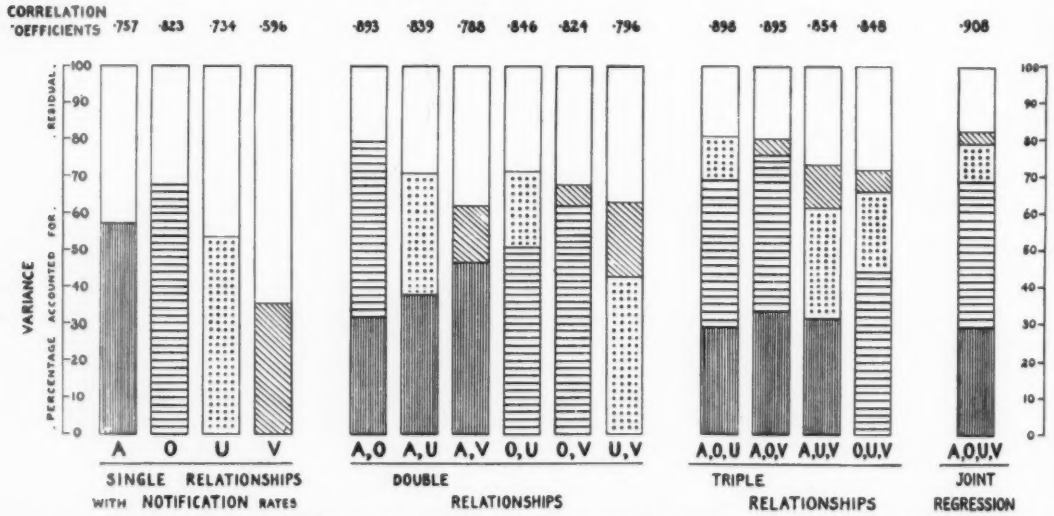


Fig. 5.—Respiratory tuberculosis notifications in the 38 wards of Glasgow, 1936–38. Contributions to variance of ward notification rates; percentages "explained" by A: ordinary crowding, O: overcrowding, U: unemployment, and V: poverty, separately and in combination.

1945–47: CORRELATIONS, REGRESSIONS, AND ANA
in relation to ORDINARY CROWDING, UN

| | SINGLE REGRESSIONS | | | | DOUBLE REGRESSION with A and U | | | |
|---|---|---|---|---|---|---|---|---|
| | Relation to | | | Inter-relation of Social Factors | Partial relation to | | Joint relation to A and U | Inter-relat |
| | A | U | R | | A | U | | |
| MORTALITY RATE: M | | | | | | | | |
| Total Correlation Coefficient .. .. | | | | | | | $R = \cdot 862$ | |
| Correlation Coefficient $r$ .. .. .. | $r_{MA} = \cdot 823$ | $r_{MU} = \cdot 677$ | $r_{MR} = \cdot 771$ | $r_{AU} = \cdot 564$ | $\cdot 726$ | $\cdot 453$ | | $r_{AU.M} =$ |
| Regression Coefficient $b$ .. .. .. | $b_{MA} = 9 \cdot 37$ | $b_{MU} = 2 \cdot 96$ | $b_{MR} = 1 \cdot 52$ | $r_{AR} = \cdot 646$ | $7 \cdot 37$ | $1 \cdot 36$ | | |
| Standard Error of Regression Coefficient .. | $1 \cdot 08$ | $\cdot 54$ | $\cdot 21$ | $r_{UR} = \cdot 780$ | $1 \cdot 18$ | $\cdot 45$ | | |
| Original Standard Deviation of Mean of M | $3 \cdot 01$ | $3 \cdot 01$ | $3 \cdot 01$ | | | | $3 \cdot 01$ | |
| Standard Deviation from Regression .. | $1 \cdot 73$ | $2 \cdot 25$ | $1 \cdot 94$ | | | | $1 \cdot 57$ | |
| "Explained" Variance .. .. .. | $67 \cdot 8\%$ | $45 \cdot 8\%$ | $59 \cdot 4\%$ | | $53 \cdot 3\%$ | $21 \cdot 1\%$ | $74 \cdot 4\%$ | |
| Variance Ratio F .. .. .. .. | $75 \cdot 7$ | $30 \cdot 4$ | $52 \cdot 7$ | | | | $50 \cdot 8$ | |
| LOG. OF MORTALITY RATE: $^{L}M$ | | | | | | | | |
| Total Correlation Coefficient .. .. | | | | | | | $R = \cdot 827$ | |
| Correlation Coefficient $r$ .. .. .. | $r_{LA} = \cdot 847$ | $r_{LU} = \cdot 665$ | $r_{LR} = \cdot 742$ | $r_{AU} = \cdot 564$ | $\cdot 766$ | $\cdot 427$ | | $r_{AU.L} =$ |
| Regression Coefficient $b$ .. .. .. | $b_{LA} = \cdot 488$ | $b_{LU} = \cdot 147$ | $b_{LR} = \cdot 074$ | $r_{AR} = \cdot 646$ | $\cdot 399$ | $\cdot 061$ | | |
| Standard Error of Regression Coefficient .. | $\cdot 051$ | $\cdot 028$ | $\cdot 011$ | $r_{UR} = \cdot 780$ | $\cdot 057$ | $\cdot 022$ | | |
| Original Standard Deviation of Mean of L | $\cdot 153$ | $\cdot 153$ | $\cdot 153$ | | | | $\cdot 153$ | |
| Standard Deviation from Regression .. | $\cdot 082$ | $\cdot 115$ | $\cdot 104$ | | | | $\cdot 075$ | |
| "Explained" Variance .. .. .. | $71 \cdot 8\%$ | $44 \cdot 2\%$ | $55 \cdot 1\%$ | | $58 \cdot 7\%$ | $18 \cdot 2\%$ | $76 \cdot 9\%$ | |
| Variance Ratio F .. .. .. .. | $91 \cdot 6$ | $28 \cdot 6$ | $44 \cdot 2$ | | | | $58 \cdot 4$ | |
| NOTIFICATION RATE: N | | | | | | | | |
| Total Correlation Coefficient .. .. | | | | | | | $R = \cdot 865$ | |
| Correlation Coefficient $r$ .. .. .. | $r_{NA} = \cdot 814$ | $r_{NU} = \cdot 786$ | $r_{NR} = \cdot 833$ | $r_{AU} = \cdot 564$ | $\cdot 664$ | $\cdot 534$ | | $r_{AU.N} =$ |
| Regression Coefficient $b$ .. .. .. | $b_{NA} = 21 \cdot 75$ | $b_{NU} = 7 \cdot 50$ | $b_{NR} = 3 \cdot 58$ | $r_{AR} = \cdot 645$ | $14 \cdot 22$ | $3 \cdot 93$ | | |
| Standard Error of Regression Coefficient .. | $2 \cdot 59$ | $\cdot 98$ | $\cdot 40$ | $r_{UR} = \cdot 780$ | $2 \cdot 70$ | $\cdot 92$ | | |
| Original Standard Deviation of Mean of N | $6 \cdot 57$ | $6 \cdot 57$ | $6 \cdot 57$ | | | | $6 \cdot 57$ | |
| Standard Deviation from Regression .. | $3 \cdot 94$ | $4 \cdot 12$ | $3 \cdot 68$ | | | | $3 \cdot 39$ | |
| "Explained" Variance .. .. .. | $65 \cdot 0\%$ | $61 \cdot 8\%$ | $69 \cdot 4\%$ | | $42 \cdot 4\%$ | $32 \cdot 4\%$ | $74 \cdot 8\%$ | |
| Variance Ratio F .. .. .. .. | $66 \cdot 86$ | $58 \cdot 3$ | $81 \cdot 7$ | | | | $52 \cdot 0$ | |

## TABLE IX

LATIONS, REGRESSIONS, AND ANALYSES OF VARIANCE OF RESPIRATORY TUBERCULOSIS RATES in relation to ORDINARY CROWDING, UNEMPLOYMENT, and POVERTY in the 38 Wards of GLASGOW.

| DOUBLE REGRESSION with A and U | | | | DOUBLE REGRESSION with A and R | | | | DOUBLE REGRESSION with U and R | | | | MULTIPLE REGRESSION | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Partial relation to | | Joint relation to A and U | Inter-relations | Partial relation to | | Joint relation to A and R | Inter-relations | Partial relation to | | Joint relation to U and R | Inter-relations | Partial relation to | | | Joint relation to A, U, R | Inter-relations |
| A | U | | | A | R | | | U | R | | | A | U | R | | |
| | | $R = \cdot 862$ | $r_{AU.M} = \cdot 017$ | | | $R = \cdot 881$ | $r_{AR.M} = \cdot 030$ | | | $R = \cdot 780$ | $r_{UR.M} = \cdot 450$ | | | | $R = \cdot 883$ | $r_{AU.MR} = \cdot 003$ |
| ·726 | ·453 | | | ·669 | ·552 | | | ·189 | ·528 | | | ·663 | ·142 | ·378 | | $r_{AR.MU} = \cdot 025$ |
| 7·37 | 1·36 | | | 6·35 | ·81 | | | ·84 | 1·22 | | | 6·22 | ·472 | ·66 | | $r_{UR.MA} = \cdot 326$ |
| 1·18 | ·45 | | | 1·19 | ·21 | | | ·74 | ·33 | | | 1·21 | ·57 | ·28 | | |
| | | 3·01 | | | | 3·01 | | | | 3·01 | | | | | 3·01 | |
| | | 1·57 | | | | 1·47 | | | | 1·94 | | | | | 1·47 | |
| 53·3% | 21·1% | 74·4% | | 46·0% | 31·6% | 77·6% | | 13·0% | 47·9% | 60·9% | | 45·0% | 7·3% | 25·7% | 78·0% | |
| | | 50·8 | | | | 60·6 | | | | 27·2 | | | | | 40·3 | |
| | | $R = \cdot 827$ | $r_{AU.L} = \cdot 001$ | | | $R = \cdot 885$ | $r_{AR.L} = \cdot 046$ | | | $R = \cdot 755$ | $r_{UR.L} = \cdot 471$ | | | | $R = \cdot 888$ | $r_{AU.LR} = -\cdot 031$ |
| ·766 | ·427 | | | ·719 | ·482 | | | ·206 | ·479 | | | ·714 | ·166 | ·295 | | $r_{AR.LU} = \cdot 056$ |
| ·399 | ·061 | | | ·364 | ·033 | | | ·049 | ·057 | | | ·356 | ·028 | ·025 | | $r_{UR.LA} = \cdot 362$ |
| ·057 | ·022 | | | ·059 | ·010 | | | ·039 | ·018 | | | ·060 | ·029 | ·014 | | |
| | | ·153 | | | | ·153 | | | | ·153 | | | | | ·153 | |
| | | ·075 | | | | ·073 | | | | ·103 | | | | | ·073 | |
| 58·7% | 18·2% | 76·9% | | 53·5% | 24·9% | 78·3% | | 14·6% | 42·4% | 57·0% | | 52·4% | 8·3% | 18·3% | 78·9% | |
| | | 58·4 | | | | 63·3 | | | | 23·2 | | | | | 42·5 | |
| | | $R = \cdot 865$ | $r_{AU.N} = -\cdot 045$ | | | $R = \cdot 875$ | $r_{AR.N} = \cdot 075$ | | | $R = \cdot 861$ | $r_{UR.N} = \cdot 365$ | | | | $R = \cdot 894$ | $r_{AU.NR} = -\cdot 078$ |
| ·664 | ·534 | | | ·642 | ·545 | | | ·395 | ·569 | | | ·574 | ·384 | ·412 | | $r_{AR.NU} = \cdot 091$ |
| 14·22 | 3·93 | | | 13·43 | 1·88 | | | 3·32 | 2·41 | | | 11·01 | 2·85 | 1·21 | | $r_{UR.NA} = \cdot 349$ |
| 2·70 | ·92 | | | 2·71 | ·43 | | | 1·31 | ·596 | | | 2·69 | 1·18 | ·46 | | |
| | | 6·57 | | | | 6·57 | | | | 6·57 | | | | | 6·57 | |
| | | 3·39 | | | | 3·27 | | | | 3·43 | | | | | 3·07 | |
| 42·4% | 32·4% | 74·8% | | 40·1% | 36·4% | 76·5% | | 27·4% | 46·8% | 74·2% | | 32·9% | 23·5% | 23·6% | 80·0% | |
| | | 52·0 | | | | 57·0 | | | | 50·3 | | | | | 45·2 | |

The actual equations give estimates of the disease rates in 1945–47 in relation to ordinary crowding, unemployment and poverty: the estimated mortality rates were these:

$$M_e = 6{\cdot}225\,A + {\cdot}472\,U + {\cdot}656\,R - 14{\cdot}083$$
$$L_e = {\cdot}356\,A + {\cdot}028\,U + {\cdot}025\,R - {\cdot}369$$

where $M_e$ represents estimated mortality rate (per 10,000 ward population).
$L_e$ represents estimated log. of mortality rate.
A represents ordinary crowding (ward average of persons per house).
U represents unemployment (adult unemployed men per cent. of population).
R represents poverty (persons on relief per cent. of ward population).

In the first equation, in which the total correlation of mortality with the social distributions was ·883, the partial correlations with ordinary crowding (·663) and with poverty (·378) are both significant. The influence of each social factor is also illustrated by each partial regression coefficient; in the case of ordinary crowding, this coefficient indicates that wards differing in average crowding by one person per house differed by more than six deaths per 10,000 persons. A similar difference of 6·225 in the death-rate would have been expected in wards which differed by 13 per cent. in the male unemployment percentage or by 9·5 per cent. in population living on relief. Since in 1945–47 the entire range of the unemployment index was only 2·4 per cent., and that of poverty 6·4 per cent., there were no ward differences in these social conditions sufficiently large to be associated with six deaths. The much greater association of ordinary crowding with mortality is evident.

The estimates of mortality obtained from the logarithmic equation given above corresponded still more closely to the actual ward rates, and in this regression the variance "explained" was 79 per cent. The parts played by the contributions of the three social variables are illustrated in Fig. 6, which analyses the mortality variance "explained" by the social distributions in simple and multiple associations. The first set of three columns depicts the variance accounted for in the simple association of mortality with each single factor; the order of importance implied by these correlations is repeated in the more complex relationships. The second set of columns demonstrates that there was some increase in "explained" variance when social distributions were combined, particularly when ordinary crowding and poverty were coupled; the combination of unemployment and poverty did not account for much more variance than did poverty alone. The final

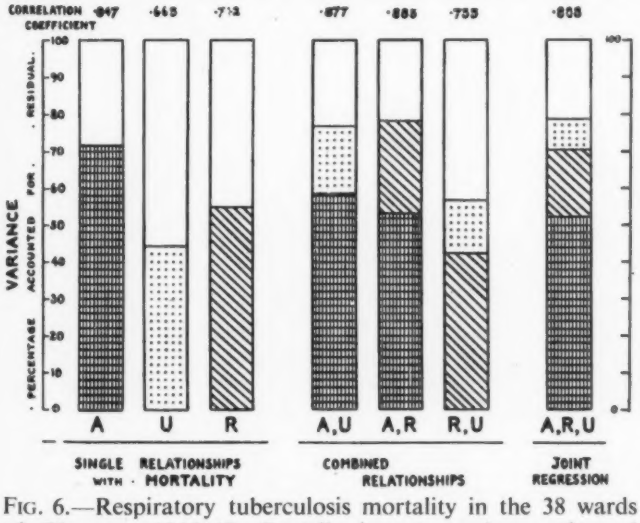


FIG. 6.—Respiratory tuberculosis mortality in the 38 wards of Glasgow, 1945–47. Contributions to variance of ward mortality rates "explained" by A: ordinary crowding, U: unemployment, and R: poverty, separately and in combination.

column illustrates the relative importance of each social component and it can be seen that the total "explained" variance was little more than that "explained" by the ordinary crowding-poverty distributions. Moreover, the contribution of ordinary crowding remained almost unchanged, but the percentage added by unemployment was nearly balanced by the reduction in the poverty contribution.

This diagram demonstrates, as does Fig. 5, that in the final regression of mortality with the social components ranked in the same order as the original simple correlation coefficients: but the magnitudes of their contributions were *not* in proportion to these simple correlations. The variance "explained" by poverty (18 per cent.) was smaller than might have been expected from the high simple correlation: and the still smaller contribution of unemployment (8 per cent.) showed a great reduction from the original 44 per cent. of mortality variance "explained" in the individual regression. It is apparent that conclusions regarding the influence of any social factor cannot be based on simple correlation coefficients alone.

The diagram shows also that the distribution of ordinary crowding did not duplicate completely the distributions of unemployment or poverty, but the latter did to some extent include within it the distribution of unemployment. If a single social variable were used as the only index of social conditions, it would not reproduce the total influence of the social complex on tuberculosis; but in individual association ordinary crowding did "explain" a very high percentage of mortality variance. But it is clearly preferable to combine at least two social distributions, and in this post-war triennium, in which no overcrowding index was considered, the combination of average crowding and poverty represented accurately the magnitude of the influence of social conditions on this disease. Moreover, it can be seen in the diagram that the residual "unexplained" variance was so small as to be of little significance in comparison with the "explained" variance; some of the total variance will always be due to chance variation, and can never be "explained" by any definite factor.

The relationship between notification distribution and social conditions in the 1945–47 triennium (as in the pre-war triennia) was not entirely like the mortality relationship; Table IX shows that the contributions of poverty and unemployment were approximately equal. Ordinary crowding, however, again played the major part in this association. The estimated ward notification rates in terms of social conditions were these:

$$N_e = 11{\cdot}013\,A + 2{\cdot}855\,U + 1{\cdot}213\,R - 20{\cdot}754$$

where $N_e$ represents estimated notification rate (per 10,000 ward population).
A represents ordinary crowding (ward average of persons per house).
U represents unemployment (adult unemployed men per cent. of population).
R represents poverty (persons on relief per cent. of ward population).

The substitution in this equation of the ward indices of ordinary crowding, unemployment, and poverty gives estimated ward notification rates which were very close to the actual ward rates.

The upper part of Fig. 7 (opposite) shows the close agreement between actual rates and regression estimates; the latter shown adjacent to the actual ward rates, and the differences between estimated and observed rates in each ward are visible. Though there were some wards in which the calculated estimate did not correspond accurately with the triennial average rate, in the great majority of wards the error of estimate was extremely small when compared with the actual ward rate or when contrasted with the original standard deviation (6·57) of the mean. The lower part of Fig. 7 shows the calculated estimates again, and illustrates the components of these estimates.

The relationships of the *ward increases* in tuberculosis rates in the years between 1936–38 and 1945–47 with the group of social factors were also examined, and a similar order of importance of the social distributions was apparent. As was expected, correlation coefficients were of a much lower order, and, though the total correlations were significant, many of the

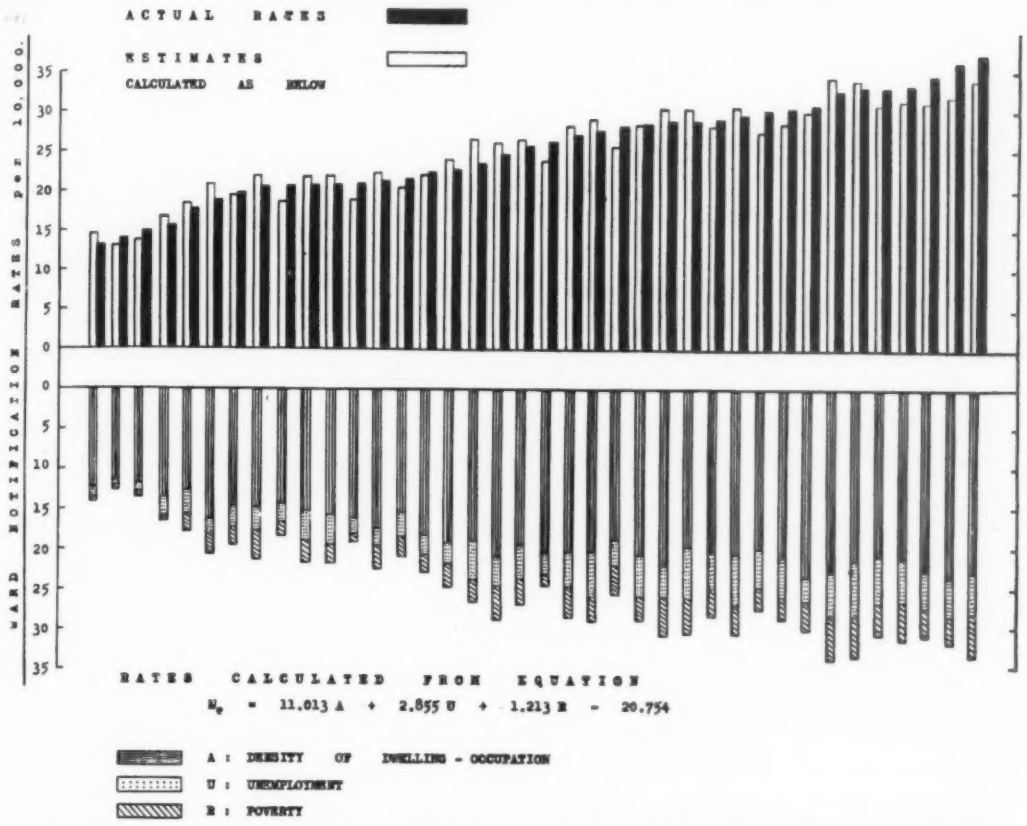
ACTUAL RATES
ESTIMATES CALCULATED AS BELOW
WARD NOTIFICATION RATES PER 10,000
RATES CALCULATED FROM EQUATION
$N_p$ = 11.013 A + 2.855 U + 1.213 R - 20.754
A : DENSITY OF DWELLING - OCCUPATION
U : UNEMPLOYMENT
R : POVERTY

FIG. 7.—Respiratory tuberculosis notifications in the 38 wards of Glasgow, 1945–47. Actual notification rates (per 10,000 population) in the separate wards, and estimated rates calculated from the regression equation.

partial correlation and regression coefficients were not significant. The multiple associations of *ward percentage increases* in the disease rates with the social complex also showed partial correlation and regression coefficients which in most cases were not significant, but which were consistently negative. Furthermore, the total correlation between the social conditions in 1936–38 and the percentage increases in notification rates (— ·51) was highly significant; and percentage mortality increases were also negatively and significantly correlated with indices of pre-war poor social conditions. Though these correlations seem small when compared with others found in the multiple regressions of mortality or notifications, they do indicate definite associations.

In general, then, the many aspects which have been examined all reiterate the significance of social conditions in relation to respiratory tuberculosis distribution. Within the group of social conditions the influence of each component and the importance of conditions of housing have become apparent. No single component can adequately represent the whole social complex; but the magnitude of its total effect in association with tuberculosis is indicated by the relationship of the disease with the combination of ordinary crowding and overcrowding. The high significance of this relationship in post-war as in pre-war years is manifest; the combination of these four

social factors "explains" so large a proportion of variance arising from ward differences in tuberculosis, that little remains to be "explained" by some unknown factor.

## Discussion

The earlier analyses in this study indicate a strong association between the distribution of respiratory tuberculosis rates and each social factor, and support the widely-held view that "tuberculosis is a social disease". They confirm also the conclusions drawn in the previous study of housing conditions and tuberculosis rates in Edinburgh (Stein, 1950). In these Glasgow analyses, however, the different magnitudes of correlations with the various social factors imply that there are real differences between the social distributions. It may be suggested that such divergencies are only the reflection of weaknesses in indices used to represent poverty and unemployment in the pre-war years. But the analyses of other mortality distributions, such as total mortality and infant mortality, and the analyses of non-respiratory tuberculosis distributions gave the expected high correlations with the indices used for poverty and unemployment; and these other mortality rates were also more highly associated with overcrowding than was respiratory tuberculosis. Hence the smaller connexions of respiratory tuberculosis with poverty and unemployment cannot be ascribed to unrepresentative indices. It has to be remembered that the relationships investigated were between *distributions* of social conditions and *distributions* of disease; and differences in the distribution of unemployment may not have as marked an effect on one disease as on another. The conclusion that respiratory tuberculosis was less strongly associated with poverty and unemployment would only seem surprising if the assumption were made that any one social index is equally representative of all social conditions. There is no basis for such an assumption. If the "social complex" *is* a complex, and separable into discrete factors, it is to be expected that these discrete components manifest their influence in individual ways. The strong connexion between respiratory tuberculosis and ordinary crowding, which is stronger for notifications in post-war years, is not to be considered as unreasonable: *ordinary* living conditions, as well as excessively bad ones, are reasonably to be expected to have considerable influence in this infectious disease.

This marked connexion, more marked when curvilinear regressions are used, leads to the obvious conclusion that notification rates are higher where the population —including the centres of infection within it—is more densely crowded. But it leads further to a more serious conclusion, that in such areas mortality increases are more than proportionally greater. This conclusion, which emerged also from the relationship of mortality to notification rates, cannot be dismissed as illusory on the grounds that in pre-war years notifications were not indicative of the true incidence of this disease. The upward curve of mortality is more marked in the post-war period; and since in these years there has been a general awareness of the need for early notification, and there have also been improvements in methods of early diagnosis, the post-war notifications should correspond more nearly with the real incidence of respiratory tuberculosis. It seems evident that the upward curve of mortality in relation to

incidence and to social conditions represents a real relationship. This may be attributable to several causes. Possibly, in the wards with high rates, new cases were notified at a later stage of the disease. Or possibly social conditions in these wards operated to speed up the advance of the disease in notified cases. Both these explanations would account for the shorter period of survival after notification which was apparent in the more crowded wards. It seems evident that, in spite of the re-housing of overcrowded tuberculous families, and in spite of treatment, hospitalization, and other methods of removing centres of infection, both the spread of infection and the further advance of the disease in the individual are accelerated in wards with disadvantageous social conditions.

Some of the findings so far discussed were previously arrived at in the Edinburgh study, in which respiratory tuberculosis was shown to be more strongly associated with conditions of housing than were other infectious diseases. In this Glasgow study the correlations of respiratory tuberculosis with the crowding distributions in the same period of time were higher and more consistent than in the Edinburgh analyses. Moreover, the similarity between the respiratory tuberculosis relationships in Glasgow and in Edinburgh was in marked contrast to the dissimilarity between the regressions of infant mortality and social conditions in the two cities. It would have been of great value to compare all infectious diseases in Glasgow with those in Edinburgh. Moreover, it is important to consider to what extent respiratory tuberculosis exhibits the same tendencies as other infectious diseases and to what extent it differs from them. It might be suggested that the relationships of non-respiratory tuberculosis rates in Glasgow with social conditions indicate some answer to this question. But it is not justifiable to base conclusions concerning infectious diseases on results found only for one, particularly as non-respiratory tuberculosis can hardly be considered as typical of the infectious diseases.

The multiple relationships investigated in the later analyses to some extent bore out the findings of the earlier simple relationships, yet revealed new aspects of these earlier results. The compound regressions threw more light on the import of the components in the social complex, and confirmed the earlier indications that the social factors did not operate completely in unison. The divergencies between the social distributions were illustrated by the manner in which the connexions of mortality or notifications with social factors were augmented when the whole group of social factors was considered. The multiple correlations were far higher than the direct correlations found in simple regressions with individual social variables. While it is to be expected that multiple correlations would give increases on the greatest of any of the single correlations, the magnitude of these increases when additional social variables were brought into the regressions was surprising. These increases demonstrated again that the overlap between the social factors is not as extensive as is often assumed, and that a single social index is not equally representative of all.

The comparison of the percentages of variance "explained" in single, double, and multiple regressions leads to a further conclusion regarding the overlap of the social distributions. The consistently large percentages of variance explained by ordinary

crowding were only slightly reduced when other social factors were included in the relationships. But the high correlation between overcrowding and respiratory tuberculosis rates in the earlier individual regressions was seen, in the multiple regressions, to include contributions of some size which were associated with poverty and unemployment. The "model" of the influence of the social complex which is obtained by using a single social index is at best not sufficiently accurate, and at worst misleading. A combination of two factors could simulate more nearly the total effect of the complex, and the ordinary crowding-overcrowding combination "explained" almost as large proportions of variance as did the whole group of factors. But no "model" can give a true picture of the relative importance of the different social factors; the addition of each index to the relationships investigated discloses more clearly the contribution of each social factor to the distribution of incidence and mortality from respiratory tuberculosis.

The suitability of the ordinary crowding-overcrowding combination as a model was thus limited, but indicated more accurately than any other pair the total variance "explained" by the whole complex. The importance of these two components was approximately equal, ordinary crowding being of more weight in mortality relationships, while overcrowding was of more weight in association with notification; each "explained" 30 to 50 per cent. of variance in one aspect of the disease and 20 to 30 per cent. of variance in the other. It is perhaps to be expected that in respiratory tuberculosis the two indices which best summarize the influence of a whole variety of social and environmental circumstances should be ordinary crowding and overcrowding. For they picture "ordinary conditions of life" and "excessively bad conditions", both important in the spread of infection and in the advance of the disease.

Though this study was concerned only with social conditions for which reliable data could be obtained, it is relevant here to consider the possible influence of other factors on the spread of respiratory tuberculosis in the general population. Certain people or groups of people are sometimes said to have a "natural tendency" towards this disease, and it is sometimes suggested that "the poor have a predisposition to tuberculosis". Those who hold such views might argue that indices of social and environmental conditions are all related to poverty, and that the present findings could still be interpreted to support their beliefs. The analyses in this study show such an argument to be fallacious: though the investigation is confined to small areas which make up the city of Glasgow, it demonstrates clearly that the contributions of different parts of the environment are of quite different importance. The residual association between the incidence of tuberculosis and poverty is of much lower order than that of other factors in the environment, and, even with the small number of social indices considered here, the weight of each contribution is different. Moreover, these variables leave a very small residuum of "unexplained" variance; and it has already been stated that *some* residuum due to chance variation must always be expected. The four social indices account for so much more of the variance than one would ordinarily expect, even if *all* factors relevant to tuberculosis (including nutrition) were taken into account, that there can be little of the variance remaining to be accounted for by

"pre-disposition". Moreover, the residual inter-relations between the social factors are much too small to have a common factor of any size. The relationships of the Glasgow ward rates to the four social distributions give consistent evidence of the different contributions made by each, and show that the "social complex" is not explicable by any single factor such as "predisposition". The contribution of predisposition, if there be any, must be exceedingly small.

One more point is worthy of mention. The data originally obtained regarding the numbers of unemployed related to the ten Employment Exchange Areas of Glasgow, and single and multiple regressions and correlations were calculated for the relationships between respiratory tuberculosis rates and crowding, overcrowding, and unemployment indices in these ten large areas. The results obtained are not published here, since they were superseded by the more detailed analyses of the 38 wards. But they led to two conclusions: firstly, though there were inconsistencies in some years, the proportions of variance "explained" were not inconsiderable; secondly, the inconsistencies indicated that the social indices were not sufficiently accurate for the areas which they represented, and that results might be more revealing if more homogeneous and smaller areas were considered. The heterogeneous character of the Employment Exchange Areas was particularly marked in the peripheral exchanges; one of these stretched from the very centre of the city to the rural area beyond the city boundary, and included within its range more than one-fifth of the population of Glasgow. For these reasons the break-down of the unemployment data for the Exchange Areas into unemployment figures for the smaller and more homogeneous wards was undertaken. The results constitute a remarkable demonstration of the value of analysing relationships involving social indices in the smallest and most exact way possible; they show how contradictory may be the results, and how doubtful the conclusions, if social conditions in a large heterogeneous area are represented by a single index. The wards on which the present study is based are still too large and insufficiently homogeneous, but no smaller administrative units exist. The more social variables are precisely measured for small areas, the more will be learned of the influence of "social conditions" on disease.

## Conclusion

The view that "social conditions" are of great importance in the spread of respiratory tuberculosis is widely held, yet it has been rather a matter of belief than of precise evidence. Precise knowledge of the total influence of "social conditions" in Scotland has not been available; and it has not been known which factors in the social complex are most important in connexion with this disease, nor what are their individual contributions to its incidence and mortality.

In this study of respiratory tuberculosis in the wards of Glasgow, the investigation of small, homogeneous areas reveals that the inequalities of incidence in this disease are very largely accounted for by disparities in social conditions. Moreover, these successive analyses with further combinations of social factors show clearly that it is possible to estimate the separate contribution of each social component to

the divergencies in respiratory tuberculosis. Other mortality is also associated with social conditions, but the factors of dominant influence are not the same as those in respiratory tuberculosis. The relationship of respiratory tuberculosis with the social complex demonstrates the important role played in Glasgow by housing conditions. Ordinary density of dwelling occupation, together with overcrowding, represents the dominant influence in infection and in death from this disease.

## Summary

(1) This study was undertaken to assess the influence of the "social complex" and its constituent factors on respiratory tuberculosis notifications and mortality in Glasgow. The importance of Glasgow rates in any investigation of Scottish tuberculosis trends is discussed. The need to acquire precise knowledge of the complex of social conditions and to disentangle its components is emphasized.

(2) Respiratory tuberculosis rates in Glasgow are described, and trends in certain age groups illustrated. Comparisons indicate that Glasgow trends have diverged from those in towns in England and Wales and in most other cities in Scotland.

(3) The scope of this study of tuberculosis in the years 1930–47 is outlined, and the indices used are stated. The statistical methods applied in the analyses (correlation, regression, and analysis of variance) are discussed. Single regressions are used in the earlier analyses, and additional variables are successively brought into multiple regressions. The reasons for such duplication are given, and the possible inadequacies of conclusions drawn from simple correlation coefficients alone are debated.

(4) The single relationships between ward respiratory tuberculosis distributions and each separate social distribution are analysed. They evidence the strong and highly significant associations of this disease with all the social variables, but show that these associations are not equal in magnitude and that some of them vary with time. The high correlations with ordinary crowding and overcrowding are greater than those with unemployment or poverty.

(5) The strong and consistent association between mortality from respiratory tuberculosis and notification rates during the same and preceding years is shown. The upward curve of mortality in relation to notifications is more marked in post-war years.

(6) The examination of the respiratory tuberculosis rates in the pre-war and post-war triennia enlarges the earlier findings of the considerable increases in mortality and notifications. The increases themselves are significantly though not strongly associated with certain pre-war social conditions in the wards, the absolute increases being greatest in the worst-housed wards. But the percentage increases in individual wards are negatively associated with poverty and overcrowding. The implication is that during the decade the spread of respiratory tuberculosis affected all areas and was relatively more disadvantageous in wards which in pre-war years enjoyed the best environmental conditions.

(7) A number of relationships of respiratory tuberculosis rates with social variables

taken two, three, and four at a time, clarify the strong and consistent connexion with the group of social factors. Total correlations in the multiple regressions are approximately 0·9, and "explained" variance is 80 per cent. or more. The proportion of "unexplained" variance, some of it due to chance variation, is exceptionally small.

The factors in the social complex are shown to contribute differing amounts to the extraordinarily high proportions of "explained" variance. The variables in the social complex do not operate as one single factor, but contribute differently to mortality and to notification variances. Ordinary crowding and overcrowding play a most important part; together they "account for" 60 to 70 per cent. of variance, and have approximately equal weight. Poverty and unemployment contribute about 5 to 15 per cent. of variance in most regressions, poverty showing a more marked connexion with mortality.

Residual inter-correlations between the social variables indicate that the distribution of ordinary crowding is not related to the other social distributions. But between these latter some overlap is apparent, and here the overcrowding distribution seems to be dominant. The conclusion is clearly indicated that the "social complex" cannot be adequately depicted by a single social index.

(8) The findings of the more complex analyses are discussed in relation to the earlier results. It becomes evident that in some cases the conclusions indicated by direct correlations in single regressions are borne out by the multiple relationships; but in other cases they are shown to have been exaggerated conclusions. The use of a "model" in representing the effect of the social complex is considered.

The view sometimes expressed that the poor have a natural pre-disposition to tuberculosis is discussed; the evidence indicates the improbability of any sizeable contribution by "pre-disposition".

(9) In order to compare respiratory tuberculosis relationships and those of other diseases, the trends of other causes of mortality are examined (see Appendix A.1). Ward distributions of total mortality, infant mortality, and non-respiratory tuberculosis in Glasgow are described. Edinburgh rates in the pre-war years are compared with those of Glasgow, and infant mortality in the two cities is contrasted.

The single relationships of the distributions of total mortality, infant mortality, and non-respiratory tuberculosis with the separate social variables are investigated (see Appendix A.2). Infant mortality and non-respiratory tuberculosis notification rates are found to be strongly associated with the social factors, particularly with poverty and unemployment. The associations of the social factors with the distributions of other mortality rates are not similar to the relationships found for respiratory tuberculosis. Ordinary crowding plays a smaller part in association with other mortality.

(10) The divergencies between the social factors require clarification, and hence social conditions in Glasgow are more fully examined.

Housing conditions are described in some detail (see Appendix B.1). Comparisons of ordinary crowding, overcrowding, and numbers of 1-roomed and 2-roomed

houses in other cities demonstrate that Glasgow housing deficiencies are so extensive as to constitute an exceptional local circumstance. Changes in sizes of houses and in densities of population in the last two decades have made only a small difference in crowding.

The trends and distributions of unemployment and poverty in Glasgow in the years 1930–47 are examined (see Appendix B.2). Ward differences in these conditions are considered, and the changing connexion between unemployment and measurable poverty is indicated.

The indices used for ordinary crowding, overcrowding, unemployment, and poverty are more fully described, and some reasons are given for the choice of pre-war and post-war poverty indices (see Appendix C).

The inter-correlations between the different social variables are discussed: though there is some overlap between certain distributions, ordinary crowding is not closely related to the other indices. It is evident that "social conditions" cannot be adequately investigated in terms of one component only.

(11) The conclusion is drawn that ordinary density of dwelling-occupation and additional overcrowding together constitute the major factors in the association of the social complex with respiratory tuberculosis in Glasgow, and play a significant part in the excess deaths from and cases of this disease in the crowded wards of the city.

I should like to express my gratitude to Professor F. A. E. Crew, whose interest and unfailing support encouraged this work. Grateful acknowledgements are also made to the Medical Officer of Health for Glasgow and to his staff, to the Controller for Scotland of the Ministry of Labour and National Service, to the Managers and Staffs of the Glasgow Employment Exchanges, and to the Scottish Controller of the National Assistance Board, for their kind co-operation and efforts in these investigations. I should like particularly to thank Dr. P. L. McKinlay and Dr. J. Bronowski for their continuing interest and valuable advice in these studies.

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## APPENDIX A.

This study has demonstrated that in the last two decades certain social factors were strongly associated with the notification and mortality rates of respiratory tuberculosis in the Glasgow wards; and that, among these social factors, ordinary crowding and overcrowding were outstanding. These associations were of such magnitude that their significance is undoubted; the likelihood of their being due to chance occurrence is less than 1 in 1,000. Yet these relationships with particular social conditions could not be considered peculiar to respiratory tuberculosis if similar associations with the same social factors were to be found for other mortality. It could be argued that the respiratory tuberculosis relationships were merely the reflection of the general association of all disease and mortality with social conditions. It is therefore desirable to carry this investigation further into the relationship borne by the social factors to different causes of mortality. These additional analyses are limited to total mortality, infant mortality, and non-respiratory tuberculosis mortality and notifications, for these were the only conditions for which ward data for the period were obtainable.

A.1. Trends of Mortality Rates in Glasgow from 1930 to 1947.—The general trends of respiratory tuberculosis in Glasgow have already been discussed. Table II showed that there was no downward direction of the rates before the war, as there had been in Edinburgh. In many wards of Glasgow the mortality and notification rates were moving upwards some years before the war.

General mortality from all causes exhibited a somewhat similar pre-war trend. The rate for the city fluctuated about an average of 14 per 1,000, and there was no indication of any consistent decline. In the first years of the war the rate rose sharply, but towards the end of the war it fell below 14, and it continued to fall after the war. Table X (overleaf) shows that this general trend was also apparent in most of the wards. In the 1930s the upper end of the range of ward rates rose slightly, reaching 20 to 23; in the post-war years only one ward rate has exceeded 17. In Edinburgh in the 1930s the mean of general mortality was slightly lower than in Glasgow, but the difference is not significant.

The trends of non-respiratory tuberculosis rates, both mortality and notifications, differed from those described above. In this disease there was a progressive decline during the 1930s. At the outbreak of war, both mortality and notifications increased, and throughout the war they continued at high levels. In the post-war years, notifications and mortality resumed their decline, but the mortality rate still remained higher than in the mid-1930s. Table X shows that disparities between wards were again evident in this disease. In pre-war years the wards at the upper end of the range had rates nearly twice as high as the city mean, and in post-war years these disparities were still as great. However, a comparison with Edinburgh rates in the 1930s reveals little difference between the cities in non-respiratory tuberculosis.

During the years 1930–37, infant mortality, like general mortality, showed little evidence of decline from its extremely high average of 100 deaths per 1,000 live births. However, in 1938 and 1939 infant mortality decreased, and, though it rose during the war, it fell subsequently; and more recently it has fallen spectacularly. In the years 1930–39 the very high city average represented an exceedingly wide range of rates; Table X shows that in some wards infant mortality was extremely low, but that in many others the rates were far higher than the city average. There were, for example, ten wards which had rates of 125 to 150 throughout almost the entire period—that is to say, with one infant death in every seven or eight live births. In the post-war period under review, the highest rates in the ward range had fallen to less than 100. Comparison with the Edinburgh figures for the 1930s shows all the Glasgow rates to be significantly higher. In the pre-war years the Glasgow average was approximately 40 per cent. higher than the Edinburgh average; and in the worst wards of

Glasgow infant mortality was 50 per cent. greater than in the worst wards of Edinburgh. At the same time, the best wards of Glasgow had rates as low as, or even lower than, the best wards of Edinburgh.

The disparities between wards and the dissimilarities between the various causes of disease and death are illustrated in another way by the coefficients of variation. In the 1930s the variability of general mortality distribution between the wards was relatively small, giving coefficients of variation between 15 and 17 per cent. In the post-war years this variability had become even less, as is shown by coefficients of variation of less than 10 per cent. On the other hand, respiratory tuberculosis and also infant mortality were much more unevenly distributed, and coefficients of variation were much higher, approximating to 30 per cent. This variability was as evident in the post-war as in the pre-war period.

The distribution of non-respiratory tuberculosis between the wards showed even greater variability. For notification rates the coefficients of variation approached 40 per cent., and for mortality rates they were well above 40 per cent.

On the whole, the trends of notification and mortality from the various causes exhibited some similarities in the pre-war period. But in the post-war years respiratory tuberculosis rates rose whilst all others declined. This is well known; yet it is of interest to observe that infant mortality, the traditional yardstick for "social conditions", though it fluctuated at a very high level throughout the pre-war years of widespread poverty and heavy unemployment, has fallen almost annually since 1944, whereas respiratory tuberculosis in the period under review has pursued a rather different course.

A.2. Mortality from Various Causes in Relation to Social Factors.—The separate relationships between each social variable and each different distribution of notifications and mortality from certain causes, in the years from 1930 to 1947, are summarized in Table XI. Here are given the coefficients of correlation of general mortality, infant mortality, and notifications of and mortality from non-respiratory tuberculosis, with ordinary crowding,

TABLE

Other Rates of Mortality and Notification

Infant Mortality Rate: per 1,000 live births. Mortality due to all causes, Non-Respiratory Tuberculosis

| Year .. .. .. .. | 1930–1 | 1931–2 | 1932–3 | 1933–4 | 1934–5 | 1935–6 |
|---|---|---|---|---|---|---|
| *Infant Mortality Rate* | | | | | | |
| Range .. .. .. .. | 21·3–151·4 | 28·8–146·1 | 26·8–147·9 | 28·5–132·7 | 44·5–156·0 | 46·7–145·1 |
| Mean Rate .. .. .. | 95·2 | 100·7 | 97·8 | 90·6 | 92·4 | 98·8 |
| Standard Deviation of Mean | 27·5 | 29·6 | 30·1 | 27·6 | 26·2 | 26·8 |
| Coefficient of Variation .. | 28·9% | 29·4% | 30·8% | 30·5% | 28·4% | 27·1% |
| *General Death Rate* | | | | | | |
| Range .. .. .. .. | 99·5–190·8 | 105·2–187·0 | 103·3–180·7 | 83·8–180·9 | 83·5–185·4 | 88·3–186·9 |
| Mean Rate .. .. .. | 141·2 | 143·6 | 140·2 | 134·7 | 136·7 | 140·9 |
| Standard Deviation of Mean | 23·6 | 22·2 | 20·9 | 21·8 | 22·5 | 22·8 |
| Coefficient of Variation .. | 16·7% | 15·5% | 14·9% | 16·1% | 16·5% | 16·2% |
| *Non-Respiratory Tuberculosis Mortality Rate* | | | | | | |
| Range .. .. .. .. | 0·67–6·39 | 0·45–4·98 | 0·84–4·69 | 0·31–4·36 | 0·26–3·97 | 0·53–4·17 |
| Mean Rate .. .. .. | 3·25 | 2·96 | 2·58 | 2·26 | 2·10 | 2·27 |
| Standard Deviation of Mean | 1·41 | 1·22 | 1·03 | 0·98 | 0·92 | 1·03 |
| Coefficient of Variation .. | 43·3% | 41·2% | 39·7% | 43·4% | 43·8% | 45·4% |
| *Non-Respiratory Tuberculosis Notification Rate* | | | | | | |
| Range .. .. .. .. | 2·07–14·88 | 1·31–14·52 | 2·01–15·00 | 1·39–10·00 | 1·34–9·94 | 0·60–10·20 |
| Mean rate .. .. .. | 9·21 | 8·80 | 7·99 | 6·60 | 6·04 | 6·07 |
| Standard Deviation of Mean | 3·59 | 3·66 | 3·44 | 2·49 | 2·27 | 2·21 |
| Coefficient of Variation .. | 39·0% | 41·6% | 43·1% | 37·7% | 37·6% | 36·4% |

overcrowding, unemployment, and poverty. Coefficients of regression are also given in cases where the correlations were significant.

Briefly, these results indicate that there was a strong connexion between infant mortality and each social factor throughout the period. Non-respiratory tuberculosis notification rates were equally strongly associated with the social indices; but mortality from this disease corresponded less closely with the social distributions, and the correspondence diminished with the years. General mortality unexpectedly revealed a consistent association in the pre-war decade with certain social factors.

In the 1930s the ward rates of general mortality corresponded most nearly with the distribution of unemployment, the correlations being highly significant. Correlations with overcrowding in this period were also significant, but there was no significant association between general mortality and ordinary crowding or poverty. In the post-war period there seemed to be a considerable change in the distribution of general mortality, and there was no connexion between it and the social variables.

Non-respiratory tuberculosis in the pre-war decade was most closely associated with the distribution of overcrowding. This disease showed a lesser connexion with ordinary crowding and unemployment in the early 1930s, but in the later 1930s its association with the distribution of unemployment increased. The correspondence of non-respiratory tuberculosis with the distribution of poverty was most marked at all times. During the two decades under review, the association of notifications with all the social variables remained very strong; but the association of mortality with the social variables lessened.

Infant mortality displayed a remarkably strong association with overcrowding, and a connexion almost equally strong with unemployment and poverty. The association with ordinary crowding was significant but of a lower order. The time-trends of these relationships were interesting; the connexion between infant mortality and ordinary crowding changed little during the period, but its connexions with unemployment and with poverty increased.

A fuller study of the correlations and regressions of general mortality with the various social

GLASGOW WARDS, 1930–47

Mortality, Non-Respiratory Tuberculosis Notifications: per 10,000 ward population (2-year moving averages).

| 1936–7 | 1937–8 | 1938–9 | 1939–40 | 1940–1 | 1944–5 | 1945–6 | 1946–7 |
|---|---|---|---|---|---|---|---|
| 6–167·5 | 37·9–151·9 | 46·0–135·6 | 36·9–145·3 | | | 30·1–106·1 | 30·2–97·5 |
| 1·1 | 91·3 | 79·6 | 82·2 | | | 65·6 | 70·6 |
| 2·2 | 26·9 | 23·1 | 24·2 | | | 19·2 | 18·5 |
| 1·9% | 29·5% | 29·0% | 29·4% | | | 29·4% | 26·2% |
| 0–192·8 | 95·1–204·2 | 94·8–203·9 | 103·7–226·4 | | | 111·1–161·5 | 115·5–168·8 |
| 4·9 | 140·3 | 134·7 | 150·9 | | | 132·4 | 136·0 |
| 3·0 | 23·6 | 23·9 | 25·1 | | | 12·8 | 13·1 |
| 5·9% | 16·8% | 17·8% | 16·6% | | | 9·7% | 9·6% |
| 3–4·22 | 0·19–4·14 | 0·0–3·85 | 1·12–6·23 | 0·85–5·69 | 0·52–6·65 | 0·72–6·12 | 0·75–5·34 |
| 2·26 | 2·24 | 2·32 | 2·73 | 3·22 | 2·71 | 2·48 | 2·29 |
| 1·07 | 0·98 | 0·91 | 1·22 | 1·26 | 1·22 | 1·17 | 0·92 |
| 7·2% | 43·8% | 39·2% | 44·7% | 39·2% | 45·1% | 47·3% | 40·1% |
| 79–9·81 | 0·82–9·92 | 1·34–9·94 | 1·40–11·09 | 1·22–12·69 | 1·38–11·08 | 1·54–9·03 | 1·18–8·34 |
| 5·91 | 5·95 | 5·58 | 5·66 | 6·41 | 5·97 | 5·11 | 4·77 |
| 2·16 | 2·24 | 2·19 | 2·40 | 2·52 | 2·35 | 1·96 | 1·78 |
| 5·6% | 37·7% | 39·3% | 42·3% | 39·4% | 39·4% | 38·3% | 37·3% |

TABLE

OTHER MORTALITY AND NOTIFICATION RATES IN RELATION TO

Correlation and Regression Coefficients of General Mortality, Infant Mortality, Non-Respiratory Tuberculosis,

| Social Index .. .. .. | | | | | | Index A: |
|---|---|---|---|---|---|---|
| Period .. .. .. .. | 1930–1 | 1931–2 | 1932–3 | 1933–4 | 1934–5 | 1935–6 |
| *General Mortality* | | | | | | |
| Correlation Coefficient *r* .. | ·307 | ·517 | ·388 | ·353 | ·362 | ·293 |
| Regression Coefficient *b* .. | 31·80 | 38·69 | 31·98 | 30·53 | 32·42 | 22·67 |
| Standard Error of *b* .. .. | 16·35 | 10·84 | 12·82 | 13·49 | 13·93 | 12·34 |
| *Infant Mortality* | | | | | | |
| Correlation Coefficient *r* .. | ·542 | ·744 | ·635 | ·610 | ·630 | ·680 |
| Regression Coefficient *b* .. | 64·11 | 74·34 | 75·26 | 67·05 | 65·64 | 61·88 |
| Standard Error of *b* .. .. | 16·81 | 11·27 | 15·49 | 14·50 | 13·50 | 11·13 |
| *Non-Respiratory Tuberculosis Mortality* | | | | | | |
| Correlation Coefficient *r* .. | ·611 | ·716 | ·546 | ·501 | ·558 | ·588 |
| Regression Coefficient *b* .. | 3·69 | 2·94 | 2·21 | 1·95 | 2·04 | 2·06 |
| Standard Error of *b* .. .. | ·808 | ·485 | ·573 | ·561 | ·507 | ·471 |
| *Non-Respiratory Tuberculosis Notifications* | | | | | | |
| Correlation Coefficient *r* .. | ·661 | ·758 | ·734 | ·700 | ·495 | ·601 |
| Regression Coefficient *b* .. | 10·20 | 9·36 | 9·96 | 6·93 | 4·47 | 4·51 |
| Standard Error of *b* .. .. | 1·96 | 1·36 | 1·56 | 1·18 | 1·31 | ·999 |

| Social Index .. .. .. | | | | | | Index U: |
|---|---|---|---|---|---|---|
| Period .. .. .. .. | 1930–1 | 1931–2 | 1932–3 | 1933–4 | | |
| *General Mortality* | | | | | | |
| Correlation Coefficient *r* .. | ·602 | ·515 | ·478 | ·546 | | |
| Regression Coefficient *b* .. | 5·73 | 3·19 | 2·49 | 3·17 | | |
| Standard Error of *b* .. .. | 1·29 | ·897 | ·775 | ·810 | | |
| *Infant Mortality* | | | | | | |
| Correlation Coefficient *r* .. | ·697 | ·628 | ·650 | ·748 | | |
| Regression Coefficient *b* .. | 7·73 | 5·18 | 4·89 | 5·51 | | |
| Standard Error of *b* .. .. | 1·34 | 1·09 | ·966 | ·815 | | |
| *Non-Respiratory Tuberculosis Mortality* | | | | | | |
| Correlation Coefficient *r* .. | ·668 | ·584 | ·641 | ·571 | | |
| Regression Coefficient *b* .. | ·378 | ·198 | ·164 | ·149 | | |
| Standard Error of *b* .. .. | ·071 | ·047 | ·033 | ·036 | | |
| *Non-Respiratory Tuberculosis Notification* | | | | | | |
| Correlation Coefficient *r* .. | ·684 | ·686 | ·671 | ·711 | | |
| Regression Coefficient *b* .. | ·988 | ·700 | ·578 | ·472 | | |
| Standard Error of *b* .. .. | ·178 | ·126 | ·108 | ·078 | | |

factors reveals a strong connexion in the 1930s with unemployment and with overcrowding. Correlations of the order of 0·5 to 0·6 were found, and regression coefficients in this period were significant at a high level. Yet in the post-war years the correlations with unemployment were only 0·1 to 0·2, and not significantly above zero. A change in the distribution of general mortality in the post-war years is similarly indicated by the correlations with ordinary crowding. In the pre-war decade small correlations of very low significance were found, but in the post-war period the correlations were zero.

XI

SOCIAL CONDITIONS IN WARDS OF GLASGOW, 1930–47

in individual relationships with Ordinary Crowding, Overcrowding, Unemployment, Poverty.

ORDINARY CROWDING

| 1936–7 | 1937–8 | 1938–9 | 1939–40 | 1941–1 | 1944–5 | 1945–6 | 1946–7 |
|---|---|---|---|---|---|---|---|
| ·384 | ·370 | ·333 | ·318 | | | —·001 | ·027 |
| 29·96 | 30·32 | 27·74 | 27·96 | | | — | — |
| 12·02 | 12·67 | 13·09 | 13·91 | | | | |
| ·728 | ·679 | ·633 | ·571 | | | ·654 | ·695 |
| 79·73 | 63·44 | 50·84 | 48·43 | | | 48·32 | 49·25 |
| 12·50 | 11·42 | 10·36 | 11·60 | | | 9·33 | 8·49 |
| ·495 | ·512 | ·572 | ·451 | ·673 | ·622 | ·558 | ·541 |
| 1·79 | 1·73 | 1·82 | 1·93 | 3·20 | 2·90 | 2·47 | 1·86 |
| ·524 | ·485 | ·433 | ·638 | ·585 | ·609 | ·612 | ·480 |
| ·728 | ·714 | ·647 | ·590 | ·700 | ·748 | ·747 | ·625 |
| 5·34 | 5·55 | 4·94 | 4·97 | 6·66 | 6·71 | 5·53 | 4·15 |
| ·838 | ·908 | ·970 | 1·13 | 1·13 | ·991 | ·818 | ·863 |

UNEMPLOYMENT

| 1936–7 | 1937–8 | 1938–9 | | | 1944–5 | 1945–6 | 1946–7 |
|---|---|---|---|---|---|---|---|
| ·633 | ·683 | ·624 | | | | ·193 | ·241 |
| 5·10 | 6·59 | 6·10 | | | | — | — |
| 1·04 | 1·17 | 1·27 | | | | | |
| ·695 | ·779 | ·741 | | | | ·670 | ·710 |
| 7·85 | 8·57 | 6·99 | | | | 18·70 | 19·07 |
| 1·35 | 1·15 | 1·05 | | | | 3·46 | 3·15 |
| ·385 | ·534 | ·702 | | | ·758 | ·750 | ·636 |
| ·144 | ·213 | ·261 | | | 1·347 | 1·275 | ·849 |
| ·057 | ·056 | ·044 | | | ·193 | ·187 | ·172 |
| ·535 | ·625 | ·676 | | | ·730 | ·640 | ·538 |
| ·405 | ·573 | ·606 | | | 2·495 | 1·818 | 1·389 |
| ·107 | ·119 | ·110 | | | ·389 | ·363 | ·363 |

Non-respiratory tuberculosis relationships were rather more consistent. Correlations with overcrowding were high, about 0·8, higher than for respiratory tuberculosis. Similar differences between non-respiratory and respiratory tuberculosis were found in connexion with poverty and unemployment. In association with ordinary crowding, however, the correlations of non-respiratory mortality were much lower, and showed considerable fluctuations within the generally-declining trend. Non-respiratory notifications were more strongly and consistently associated with ordinary crowding, although the correlations were still less than those in the respiratory tuberculosis relationships.

TABLE XI—*cont.*

| Social Index | Index P: 1931 (Persons per room) | | Index T: 1931 (Percentage living 2+ per room) | | Index O: 1935 (Percentage of houses overcrowded) | |
|---|---|---|---|---|---|---|
| Period | 1930–1 | 1931–2 | 1930–1 | 1931–2 | 1934–5 | 1935–6 |
| *General Mortality* | | | | | | |
| Correlation Coefficient *r* | ·544 | ·521 | ·587 | ·574 | ·511 | ·466 |
| Regression Coefficient *b* | 28·38 | 25·55 | ·767 | ·705 | ·822 | ·759 |
| Standard Error of *b* | 7·41 | 7·08 | ·179 | ·170 | ·231 | ·240 |
| *Infant Mortality* | | | | | | |
| Correlation Coefficient *r* | ·807 | ·792 | ·825 | ·832 | ·738 | ·638 |
| Regression Coefficient *b* | 49·01 | 51·85 | 1·25 | 1·36 | 1·38 | 1·22 |
| Standard Error of *b* | 6·07 | 6·75 | ·145 | ·154 | ·210 | ·246 |
| *Non-Respiratory Tuberculosis Mortality* | | | | | | |
| Correlation Coefficient *r* | ·757 | ·705 | ·784 | ·710 | ·715 | ·743 |
| Regression Coefficient *b* | 2·35 | 1·90 | ·061 | ·048 | ·047 | ·055 |
| Standard Error of *b* | ·343 | ·323 | ·0081 | ·0080 | ·0077 | ·008 |
| *Non-Respiratory Tuberculosis Notification* | | | | | | |
| Correlation Coefficient *r* | ·815 | ·806 | ·851 | ·839 | ·811 | ·745 |
| Regression Coefficient *b* | 6·46 | 6·52 | ·169 | ·170 | ·131 | ·117 |
| Standard Error of *b* | ·778 | ·809 | ·018 | ·019 | ·016 | ·018 |

The connexions between infant mortality and the various social indices were all strong. Correlation coefficients with overcrowding and unemployment were much higher than those obtained for respiratory tuberculosis. Infant mortality correlations with unemployment were consistently in the region of 0·7. Regression coefficients indicate that, in the early 1930s, wards differing by 1 in the unemployment index (about one-twelfth of the range) differed by more than five infant deaths per 1,000 live births. In the post-war period, wards differing by 0·2 in the unemployment index (again, about one-twelfth of the post-war unemployment range) differed by approximately four infant deaths per 1,000 live births: though this indicated some improvement, the differences in infant mortality concomitant with social inequalities were marked. Poverty was much more strongly and consistently associated with infant mortality throughout the entire period than with any other mortality distribution investigated. Infant mortality was not so consistently associated with ordinary crowding; in the early 1930s, the correlations were not high, and later in the pre-war decade they became higher. In the post-war years, however, the correspondence between infant mortality and ordinary crowding diminished again.

Regression equations again disclosed the great influence of social conditions on infant mortality. It appeared more marked in Glasgow than in Edinburgh in the pre-war decade. Some of the regression coefficients are translated below to illustrate this comparison of pre-war infant mortality differences in the two cities.

| Difference between Ward Social Conditions | | Difference in Infant Mortality Rate |
|---|---|---|
| Wards differing by 5 per cent. in percentage of overcrowded houses | Glasgow | 7 deaths per 1,000 live births |
| | Edinburgh | 3 to 3·5 deaths per 1,000 live births |
| Wards differing in ordinary crowding by 1 person per house | Glasgow | 60 to 75 deaths per 1,000 live births |
| | Edinburgh | 20 to 25 deaths per 1,000 live births |

In the post-war years, no regressions for Edinburgh were available. But in Glasgow, in the post-war years, wards having on an average a difference of one person per house differed by nearly fifty infant

| Index V (Valuation, 1931) | | Index V (Valuation, 1936) | | | Index R (Poor Relief, 1945–6) | | |
|---|---|---|---|---|---|---|---|
| 1930–1 | 1931–2 | 1934–5 | 1935–6 | | 1944–5 | 1945–6 | 1946–7 |
| ·332 | ·316 | ·199 | ·158 | | | ·158 | ·193 |
| ·756 | ·677 | | | | | | |
| ·363 | ·344 | | | | | | |
| ·726 | ·695 | ·486 | ·372 | | | ·767 | ·775 |
| 1·925 | 1·984 | 1·235 | ·968 | | | 9·647 | 9·363 |
| ·308 | ·347 | ·370 | ·209 | | | 1·343 | 1·272 |
| ·646 | ·605 | ·405 | ·591 | | ·825 | ·819 | ·778 |
| ·0874 | ·0711 | ·0362 | ·0527 | | ·660 | ·626 | ·468 |
| ·0175 | ·0158 | ·0136 | ·0141 | | ·0754 | ·0731 | ·0629 |
| ·658 | ·645 | ·528 | ·607 | | ·892 | ·811 | ·743 |
| ·228 | ·228 | ·1302 | ·1300 | | 1·371 | 1·036 | ·863 |
| ·0440 | ·0456 | ·0296 | ·0283 | | ·116 | ·125 | ·130 |

deaths per 1,000 live births. It can be seen that the great divergence between infant mortality in the two cities was especially marked in the more crowded wards.

Thus it is evident that differences in social conditions were strongly but not equally operative in mortality and notifications from various causes. Respiratory tuberculosis was more strongly associated with factors which had relatively smaller influence in other mortality, and poverty and unemployment were of greater importance in their connexions with infant mortality and non-respiratory tuberculosis notifications. The social complex is again shown to have different effects upon different diseases.

## APPENDIX B.

The diversity of the associations found between the various causes of death or disease and the components of the social constellation has been demonstrated repeatedly in the foregoing analyses. It is apparent that other distributions of mortality do not resemble respiratory tuberculosis in its exceptionally strong association with conditions of housing. It is of interest, therefore, to study more fully these social circumstances which bear so strongly and yet differently on the distributions of disease. The following paragraphs describe social circumstances in Glasgow, and particularly conditions of housing, in the years from 1930 to 1947, and go on to analyse more fully the ward distributions and the inter-relations of these components of the social complex.

B.1. Housing in Glasgow.—The peculiarities and shortcomings of Scottish housing in general are especially evident in Glasgow. Here the peculiarities lie in the large proportion of one-roomed and two-roomed dwellings, the great quantity of old tenement housing, and the resultant density of houses and persons per acre. The shortcomings are apparent in the lack of amenities in these small "houses"*, in the disrepair and insanitary conditions, and in the overcrowding superimposed on generally-crowded conditions. These conditions in

* A house in Scotland is a self-contained dwelling with its own entrance.

themselves are possibly worse than those in the slums of other cities; but their exceptional character is due not so much to the nature of the deficiencies as to their great quantity. The extent to which these circumstances prevail is not commonly known; the "housing problem" in Glasgow is not confined, as in other cities, to a few areas forming only a small proportion of the total, but is acute in almost all regions of the city. Overcrowding is generally regarded as excessively serious if the official figure approaches 5 per cent.; thus it is difficult to imagine a picture of "slum" housing exceeding 50 per cent., of houses condemned as unfit being nearly 8 per cent., and of official overcrowding figures approaching 40 per cent. Yet that was the picture in Glasgow, and post-war building has so far made no radical change. The comparison shown in Table XII, of pre-war houses in Glasgow and in some English towns, was made recently by the Medical Officer of Health for Glasgow (1949b).

TABLE XII

OCCUPIED HOUSES OF DIFFERENT SIZES, 1931

Percentage distribution according to number of rooms

| Number of Rooms in House | | 1 | 2 | Total 1 or 2 | 3 | 4 | Total 3 or 4 | 5 or more |
|---|---|---|---|---|---|---|---|---|
| Percentage of Houses of this size | Glasgow | 14·8 | 43·7 | 58·5 | 23·7 | 9·1 | 32·8 | 8·7 |
| | Liverpool | 0·3 | 2·7 | 3·0 | 8·7 | 21·7 | 31·4 | 65·6 |
| | Manchester | 0·2 | 1·7 | 1·9 | 10·0 | 39·9 | 49·9 | 48·2 |

OVERCROWDED HOUSES, 1935

Percentage of houses of £45 or less rental found overcrowded, according to the standard of the 1935 Acts.

GLASGOW, 31·2 per cent. LIVERPOOL, 7·4 per cent. MANCHESTER, 2·1 per cent.

Similarly, the data on population density disclosed by the Census Reports and the Overcrowding Survey is illustrated in Table XIII.

TABLE XIII

DENSITY OF POPULATION, 1931–49

Persons per Acre, according to Census and Survey Reports

| Year | 1931 | 1935 | 1938 | 1945 | 1947 | 1949 |
|---|---|---|---|---|---|---|
| England and Wales | 1·1 | | | | | |
| Greater London | 18·5 | | | | | |
| County boroughs | 17·1 | | | | | |
| Other urban areas | 3·3 | | | | | |
| Rural districts | 0·2 | | | | | |
| Glasgow | 36·9 | 37·4 | 28·4* | 26·4 | 27·7 | 27·9 |
| Edinburgh | 13·6 | 14·2 | 14·4 | 13·1 | 14·9 | 14·7* |

* Extension of city boundaries, taking in more acreage, took place in Glasgow in 1938, and Edinburgh 1949.

The actual magnitude of the Glasgow problem is seen in the more recent figures shown in Table XIV (opposite); the improvements of the last 20 years have made only a small difference to the numbers of occupied one-roomed and two-roomed houses.

TABLE XIV

OCCUPIED HOUSES OF DIFFERENT SIZES, 1935–49

Percentage distribution, according to rooms: and actual numbers of 1-roomed and 2-roomed houses

| Number of Rooms in House .. .. | | | | 1 | 2 | 3 | 4 | 5 or more | Total |
|---|---|---|---|---|---|---|---|---|---|
| Houses of this Type | Glasgow | 1935* | Per cent. of total | 13·5 | 43·5 | 27·9 | 11·5 | (3·6) | 100 |
| | | | Actual number | 34,700 | 112,200 | | | | 257,600 |
| | | 1945 | Per cent. of total | 12·8 | 38·4 | 27·5 | 13·2 | 8·1 | 100 |
| | | | Actual number | 37,000 | 111,000 | | | | 289,000 |
| | | 1949 | Per cent. of total | 12·1 | 37·2 | 28·1 | 14·6 | 8·0 | 100 |
| | | | Actual number | 35,900 | 110,400 | | | | 296,400 |
| | Edinburgh | 1946 | Per cent. of total | 3·9 | 25·4 | 32·3 | 20·3 | 18·1 | 100 |
| | | | Actual number | | | | | | 120,300 |

* The numbers for 1935 relate to houses of £45 or less rental.

In 1944 the Medical Officer of Health for Glasgow estimated the city's housing needs for replacing unfit houses, relieving overcrowding, and fulfilling general needs (Glasgow, 1943). His estimate totalled 100,000 new houses, overcrowding being assessed by the old standard of the Housing (Scotland) Act of 1935. His further estimate, if the 1944 standard of occupancy used in England and Wales were adopted, showed that 158,000 new houses were needed: of these, 108,000 were required to relieve overcrowding alone. At that time the total number of occupied houses in Glasgow was less than 290,000. Thus the extent of overcrowding in the city, even by statutory standards, is manifest.

These tables show only the average figures for the city taken as a whole. Many wards had conditions much below the average in every respect, and Table XV illustrates the range of population densities in the wards. Similar ward variations exist in overcrowding and in

TABLE XV

DENSITY OF POPULATION IN WARDS OF GLASGOW

Pre-war and post-war distribution by Persons per Acre

| Year | Persons per Acre | 0–10 | 11–30 | 31–40 | 41–50 | 51–70 | 71–100 | 101–130 | 130+ | Persons per Acre | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | | | | Whole City | Densest Ward |
| 1934 | Number of Wards in each Range | 2 | 8 | 2 | 3 | 6 | 7 | 6 | 4 | 37 | 190 |
| 1938* | | 3 | 8 | 2 | 1 | 7 | 9 | 4 | 4 | 28 | 188 |
| 1945 | | 4 | 8 | 1 | 5 | 4 | 9 | 3 | 4 | 26 | 168 |
| 1947 | | 3 | 8 | 2 | 5 | 4 | 9 | 3 | 4 | 28 | 174 |

* The extension of city boundaries, taking in more acreage, took place in Glasgow in 1938.

general crowding. In the 1930s the old standard of overcrowding (three "persons"* per room) was abandoned in Scotland as being too harsh, and overcrowding was recognized if the occupancy exceeded two "persons" per room. Yet in Glasgow the 1931 Census disclosed that over 42 per cent. of the population were living more than two per room; moreover, 19 per cent. were living more than three per room, and 7½ per cent. more than four per room. Table XVI (overleaf) shows that these conditions, though worse in some wards than in others, were not confined to a few areas, but were general in the great majority.

Such housing conditions are quite exceptional, even in Scotland where the nineteenth century has left this legacy of one- and two-roomed dwellings located in tenements. For example, though the Edinburgh Survey of 1946 revealed that nearly 14 per cent. of all families

* Children aged 1 to 9 years count only as "half persons", and infants under 1 year are not counted at all.

TABLE XVI

DISTRIBUTION OF POPULATION IN THE WARDS OF GLASGOW, 1931

Percentages living in 1-roomed and 2-roomed houses and living 3+ per room or 4+ per room

| Percentage of ward population living in 1-roomed or 2-roomed houses .. .. | Under 10 | 10–19 | 20–29 | 30–49 | 50–69 | 70+ |
|---|---|---|---|---|---|---|
| Number of wards .. .. .. .. | 2 | 3 | 1 | 7 | 15 | 9 |
| Percentage of ward population living 3+ per room .. .. .. .. .. | Under 5 | 5–9 | 10–14 | 15–19 | 20–29 | 30+ |
| Number of wards .. .. .. .. | 6 | 3 | 2 | 10 | 12 | 4 |
| Percentage of ward population living 4+ per room .. .. .. .. .. | Under 5 | 5–9 | 10–14 | 15–19 | 20–29 | 30+ |
| Number of wards .. .. .. .. | 10 | 20 | 5 | 2 | — | — |

were statutorily overcrowded by Scottish standards, and nearly 33 per cent. by English standards, Edinburgh compares most favourably with Glasgow. Table XVII (pp. 44-45) shows the ranges and means of Glasgow ward housing rates and other social indices from 1930 to 1948. The general crowding (average number of persons per house)* changed little in the pre-war decade, the mean falling from 4·05 to 3·86.

By contrast, the mean of the ward averages in Edinburgh fell from 3·93 to 3·65, and the statutory overcrowding figures for 1931 and for 1935 similarly demonstrate that dwelling-occupation was considerably more dense in Glasgow than in Edinburgh. In 1931 the Glasgow ward rates gave averages of 1·63 persons per room and more than 40 per cent. living 2+ per room, and in 1935 over 29 per cent. of houses were overcrowded.† In Edinburgh in 1931 there were 1·19 persons per room and 27 per cent. living 2+ per room, and in 1935 17 per cent. of houses were overcrowded.

This long and detailed description of certain aspects of Glasgow housing indicates the possible part played in the spread of infectious disease by such exceptional local circumstances; their relative influence seems to be much greater in respiratory tuberculosis than other diseases or in other localities.

B.2. LEVELS OF UNEMPLOYMENT AND POVERTY IN GLASGOW.—The extent of male unemployment in the city has changed considerably during the last 20 years, first as the depression of 1930 began to reach local industry, then as its effects were felt in other occupations, and subsequently as the periods of partial recovery, of war, and of post-war full employment followed upon each other. In the "good" period of 1927–29 the city average was three unemployed adult men per 100 persons. In 1930 and 1931 this rate rose steeply. Early in 1933 the peak was reached: in every 100 persons of all ages, nine men aged 21–64, one younger man, and two women were *registered* unemployed. When one remembers those not registered and those not insurable, and considers also the wives, children, and dependants of the twelve registered, it is manifest that a large percentage of the population was directly affected by unemployment. The high rates continued for some years and then fell slowly, but even in the years immediately before the war the unemployed adult men numbered nearly 5 per cent. of the population. In recent years the rate has been much lower, approximately 1 per cent. of the population.

* "Persons per house" rather than "persons per room" is used as a measure of general crowding, since the small Glasgow houses have only one kitchen-living room for all general purposes.

† In the worst wards over 65 per cent. of the people lived 2+ per room and 54 per cent. of houses were overcrowded.

In different wards the proportions of unemployed varied widely from the city average (Table XVII). Early in 1931 the unemployed adult men numbered 15 per cent. of the population in some wards, which implies that in these wards 40 per cent. to 50 per cent. of the population were directly affected. For several years there were nine wards with rates of 12 or more per cent. Even in 1938 there were some wards with rates as high as the peak city rates during the depression.

The changes in the numbers living in poverty were also particularly evident in the mid-1930s, as more and more people exhausted their unemployment insurance. There was a time-lag between the changes in employment levels and the numbers dependent on relief, and though the number of able-bodied unemployed receiving assistance began to lessen after 1936, those on ordinary poor relief continued to increase. At the beginning of the decade the heads of household receiving relief on the Ordinary Outdoor and Able-bodied Rolls averaged 17,000 and 8,000 respectively. From 1934 until 1936 the unemployed heads of households on assistance were always between 26,000 and 29,000, but in 1937 they fell greatly. The numbers of "ordinary poor" continued to mount sharply until 1935, when heads of households alone totalled 40,000, and from then until the outbreak of war they remained at or above this level. In 1940 the heads of households on the Ordinary Roll fell to 19,000, and in the following years diminished very slowly. Thus the effects of the depression years were reflected first in the numbers of "able-bodied" on relief, which reached a peak from 1934 to 1936, and more gradually in the "ordinary poor", who reached their highest numbers from 1935 to 1939.

The applicants for assistance, together with their dependants, varied in the different wards, but numbers were high even during the war. From 1945 to 1947 the total numbers chargeable on the Outdoor Rolls averaged 3 per cent. of the city population. Some wards had less than 1 per cent. but seven wards had more than 5 per cent. of their population depending on relief.

The disparities in social and environmental circumstances in the different wards are further illustrated by a comparison in Table XVII of the coefficients of variation for the different indices.* The variability in the distribution of general crowding (as measured by average persons per house) was small, the standard deviation being consistently from 6 to 8 per cent. of the mean. But the variability in ward rates of persons per room in 1931 was much greater; and the distribution of overcrowding both in 1931 and in 1935 showed still greater variation, with coefficients nearing 50 per cent. The distribution of unemployment throughout the whole period also varied greatly, with coefficients comparable to those of overcrowding; as did the distribution of poverty in 1945–47. The great variability of ward valuation rates, with standard deviations nearly double the means, reflects the character of the actual distribution; four well-off wards, with extremely high valuation, stand out against fourteen very poor wards with almost zero valuation and six others with extremely low rates. It has to be remembered that "valuation" is in reality an index of wealth rather than of poverty.

B.3. Changes and Inter-Relations in the Social Distributions.—Changes in the different social distributions are evidenced by the correlations between the same index in different years (Table XVIII). The high correlations of ordinary crowding show that, though average numbers per house lessened slightly with the years, the distribution of crowding among the wards changed little. Similarly, the overcrowding distributions were highly correlated with each other, indicating that the relative inequalities between the wards remained almost the same. These indices, however, relate only to the 1930s, and since no overcrowding data for more recent times are available, no conclusion can be drawn about the present distribution of overcrowding. The distribution of unemployment in the different wards changed slightly

* The indices used and the reason for their selection are described in Appendix C.

TABLE

SOCIAL INDICES IN WARDS

ORDINARY CROWDING, UNEMPLOYMENT,

A: Ordinary Crowding: Density of dwelling-occupation (average persons per house in each ward).
U: Unemployment: Adult males registered as unemployed, per cent. of total population in each ward.
P: Overcrowding: Density of room-occupation—average persons per room in each ward.
T: Excess room-crowding—percentage of ward population living 2+ per room.

| Year | 1930 | 1931 | 1932 | 1933 | 1934 | 1935 | 1936 |
|---|---|---|---|---|---|---|---|
| *Persons per House:* A | | | | | | | |
| Range | 3·46–4·46 | 3·32–4·47 | 3·36–4·15 | 3·37–4·46 | 3·37–4·46 | 3·28–4·51 | 3·28–4·51 |
| Mean | 4·05 | 4·02 | 4·02 | 4·02 | 4·02 | 3·99 | 3·99 |
| Standard Deviation of Mean | ·233 | ·297 | ·254 | ·251 | ·251 | ·295 | ·295 |
| Coefficient of Variation | 5·8% | 7·4% | 6·3% | 6·3% | 6·3% | 7·4% | 7·4% |
| *Unemployment:* U | | | | | | | |
| Range | 0·9–9·9 | 1·4–14·9 | 1·5–15·0 | 1·3–14·4 | | | 0·9–10·7 |
| Mean | 5·0 | 7·5 | 8·4 | 7·9 | | | 5·5 |
| Standard Deviation of Mean | 2·5 | 3·6 | 4·0 | 3·8 | | | 2·9 |
| Coefficient of Variation | 49·3% | 47·8% | 47·5% | 47·6% | | | 52·4% |

| | P. 1931 Persons per room | T. 1931 Percentage living 2 + per room | O. 1935 Percentage houses overcrowded |
|---|---|---|---|
| *Overcrowding:* P, T, O | | | |
| Range | 0·65–2·47 | 1·8–69·3 | 1·66–54·35 |
| Mean | 1·63 | 40·4 | 29·2 |
| Standard Deviation of Mean | 0·453 | 18·10 | 14·01 |
| Coefficient of Variation | 27·7% | 44·8% | 48·1% |

but noticeably in the later years of the period investigated. During the early 1930s it was unaltered, as is shown by the correlations of 0·99 in adjacent years; by 1936–38 it was somewhat differently distributed between the wards, and gave a lower correlation with the 1930–33 distribution.

The relationships between the social factors are indicated by the inter-correlations between the different indices (Table XVIII, overleaf). The associations may be described as follows:

*Ordinary Crowding and Overcrowding*, stronger in 1931 than in 1935, but not high; correlation coefficients fell from about 0·7 to about 0·6.

*Ordinary Crowding and Unemployment* not high; correlation coefficients in the early 1930s slightly above 0·6; in the later 1930s and after the war somewhat lower.

*Poverty and Ordinary Crowding* rose slightly; pre-war correlation coefficients 0·55, and post-war 0·65.

*Unemployment and Overcrowding* strong in the 1930s; the correlation coefficients mostly well above 0·7.

*Unemployment and Poverty*, slight in the 1930s; correlation coefficients only just above 0·4. After the war closely allied; correlation coefficient of 0·8 higher than any previously mentioned.

*Poverty and Overcrowding*, in the 1930s comparatively high; correlation coefficients of about 0·8.

These relationships demonstrate that, though each social factor is to some extent associated with others, the social variables are by no means identical. The overlap between some of them is small, and they stand in different relation at different times. Ordinary crowding was not distributed in the same way as unemployment and poverty; and unemployment in the

XVII

OF GLASGOW, 1930–1948

OVERCROWDING, and POVERTY

O: Statutory overcrowding—percentage of houses in each ward found to be overcrowded.
V: Poverty: Valuation of district—proportion of houses over £45 rental in relation to other rentals in ward.
R: Outdoor Relief—percentage of ward population on Outdoor Rolls chargeable to Welfare Department.

| 1937 | 1938 | 1939 | 1940 | 1944 | 1945 | 1946 | 1947 | 1948 |
|---|---|---|---|---|---|---|---|---|
| 3·21–4·42 | 3·21–4·30 | 3·19–4·40 | 2·93–4·03 | 2·89–3·98 | 2·89–3·97 | 2·95–4·01 | 3·00–4·14 | 3·23–4·54 |
| 3·91 | 3·89 | 3·86 | 3·56 | 3·51 | 3·51 | 3·59 | 3·65 | 3·69 |
| ·289 | ·287 | ·285 | ·265 | ·262 | ·265 | ·268 | ·273 | ·301 |
| 7·4% | 7·4% | 7·4% | 7·5% | 7·5% | 7·5% | 7·5% | 7·5% | 8·1% |
| | 0·7–8·9 | | | | 0·4–4·8 | | 0·2–2·6 | |
| | 4·5 | | | | 2·5 | | 1·4 | |
| | 2·4 | | | | 1·3 | | 0·7 | |
| | 54·2% | | | | 51·3% | | 50·7% | |

| | V. 1931–2 Valuation | V. 1935–6 Valuation | R. 1946 Outdoor Relief |
|---|---|---|---|
| *Poverty:* V | | | |
| Range .. .. .. | 0·08–54·02 | 0·09–53·83 | 0·35–6·74 |
| Mean .. .. .. | 6·44 | 6·57 | 2·99 |
| Standard Deviation of Mean | 10·58 | 10·32 | 1·53 |
| Coefficient of Variation .. | 164·3% | 156·9% | 51·2% |

depression years of 1930–34 was not distributed in the same way as in 1946–47. Moreover, the associations found in Glasgow indicate that the connection of ordinary crowding with poverty and unemployment is much smaller than is sometimes assumed. Thus the same conclusion is reached as in the multiple analyses of respiratory tuberculosis in relation to the social complex, that one social index is not equally representative of all, and that "social conditions" cannot be adequately investigated in terms of one component only.

## APPENDIX C.

C.1. INDICES USED.—The disease indices used in the preceding analyses are ward rates of mortality and ward rates of notification. The social indices are average densities of dwelling-occupation and of room-occupation, percentages of *population* having excessive room-crowding, percentages of *houses* statutorily overcrowded, male unemployment rates relative to total population, ward valuation according to rentals, and poverty indicated by proportions of population on relief. All these rates, percentages, and averages refer to the individual wards of Glasgow, and the means used are the means of the ward rates, not the averages for the city taken as a whole.

*Disease Rates.*—Mortality and notification rates are calculated as 2-year moving averages; in addition, for the pre-war and post-war comparison, triennial averages are used. For most causes, the rates refer to the total deaths or cases per 10,000 population, but infant mortality is reckoned in the usual way per 1,000 live births. Logarithms of mortality rates are to the base 10.

*Crowding.*—The average density of dwelling-occupation, the index used as a measure

## TABLE XVIII

INTER-RELATIONS BETWEEN SOCIAL VARIABLES IN WARDS OF GLASGOW, 1930–47

Ordinary Crowding, Overcrowding, Unemployment, and Poverty

*(a) Correlations of each social variable at different periods*

| ORDINARY CROWDING: A | | | | UNEMPLOYMENT: U | | | |
|---|---|---|---|---|---|---|---|
| Years | | | Correlation | Years | | | Correlation |
| A 1935 | A 1945 | | ·991 | U 1930 | U 1931 | | ·989 |
| A 1936 | A 1946 | | ·987 | U 1930 | U 1932 | | ·987 |
| A 1937 | A 1947 | | ·994 | U 1931 | U 1932 | | ·990 |
| | | | | U 1931 | U 1933 | | ·986 |
| | | | | U 1932 | U 1933 | | ·992 |
| OVERCROWDING: P, T, O | | | | U 1932 | U 1936 | | ·894 |
| | | | | U 1933 | U 1936 | | ·887 |
| P 1931 | T 1931 | | ·980 | U 1933 | U 1938 | | ·868 |
| P 1931 | O 1935 | | ·987 | U 1936 | U 1938 | | ·929 |
| T 1931 | O 1935 | | ·992 | | | | |
| | | | | U 1936 | U 1945 | | ·871 |
| | | | | U 1938 | U 1945 | | ·881 |
| POVERTY: V, R | | | | U 1938 | U 1947 | | ·877 |
| | | | | U 1933 | U 1945 | | ·855 |
| V 1931 | V 1935 | | ·964 | U 1933 | U 1947 | | ·834 |
| V 1931 | R 1946 | | ·784 | | | | |
| V 1935 | R 1946 | | ·816 | U 1945 | U 1947 | | ·922 |

*(b) Inter-correlations between different variables*

| ORDINARY CROWDING AND OVERCROWDING, UNEMPLOYMENT, POVERTY | | | | UNEMPLOYMENT AND OVERCROWDING, POVERTY | | | |
|---|---|---|---|---|---|---|---|
| Variables | | | Correlation | Variables | | | Correlation |
| A 1931 | P 1931 | | ·695 | U 1930 | P 1931 | | ·741 |
| A 1931 | T 1931 | | ·731 | U 1930 | T 1931 | | ·757 |
| A 1931 | O 1935 | | ·548 | U 1931 | P 1931 | | ·683 |
| A 1935 | T 1931 | | ·622 | U 1931 | T 1931 | | ·720 |
| A 1935 | O 1935 | | ·578 | U 1932 | P 1931 | | ·623 |
| | | | | U 1932 | T 1931 | | ·701 |
| A 1930 | U 1930 | | ·546 | U 1936 | O 1935 | | ·728 |
| A 1931 | U 1931 | | ·640 | U 1938 | O 1935 | | ·714 |
| A 1932 | U 1932 | | ·612 | U 1947 | O 1935 | | ·775 |
| A 1933 | U 1933 | | ·631 | | | | |
| A 1936 | U 1936 | | ·579 | U 1931 | V 1931 | | ·571 |
| A 1938 | U 1938 | | ·570 | U 1936 | V 1935 | | ·435 |
| A 1945 | U 1945 | | ·573 | U 1938 | V 1935 | | ·409 |
| A 1947 | U 1947 | | ·562 | U 1947 | V 1935 | | ·463 |
| | | | | U 1945 | R 1946 | | ·812 |
| A 1931 | V 1931 | | ·555 | U 1947 | R 1947 | | ·780 |
| A 1935 | V 1935 | | ·547 | | | | |
| A 1946 | R 1946 | | ·655 | OVERCROWDING AND POVERTY | | | |
| A 1947 | R 1947 | | ·646 | | | | |
| | | | | P 1931 | V 1931 | | ·787 |
| | | | | T 1931 | V 1931 | | ·763 |
| | | | | O 1935 | V 1935 | | ·709 |

of ordinary crowding, is calculated for each year from 1930 to 1947 in each ward as the average number of persons of all ages per house.* For 1930–32 and 1935–38 indices of overcrowding as well as of ordinary crowding are used. In the 1930–32 period, two overcrowding indices are obtainable: the first is density of room-occupation, calculated as the average number of persons per room, and the second is excess room-crowding, calculated as the percentage of the ward *population* living more than two persons per room. In the 1935–38 period one index, based on statutory overcrowding as revealed by the 1935 Survey, is considered; ward rates are calculated according to the percentages of *houses* found to be overcrowded according to the Scottish standard.

*Unemployment.*—These rates are based on the numbers of insured adult males in each ward who registered as unemployed in the summer months in each year, and are calculated as percentages of entire ward populations. Various indices to represent the distribution of unemployment in the wards were considered, and the rates used were limited to those for adult males for three periods: the "depression" years of the early 1930s, the "recovery" years of the late 1930s, and the post-war "full employment" period.

The limitations were adopted for the following reasons:

Firstly only summer unemployment has been taken into account in order to minimize the effects of seasonal interruptions of work.

Secondly, a high correlation was found between the different unemployment distributions in the years 1930, 1931, 1932, and 1933; it was therefore thought preferable to use one set of rates for this period rather than the separate sets for each of the years. High correlations found in the immediate pre-war years and again in the post-war period also led to the use of a single set of rates for each period.

Thirdly, female and juvenile unemployment have been excluded in arriving at the index, since both fluctuate in other ways and for other reasons than does the unemployment of the chief wage-earners. This is particularly evident in the depression years, in which the extractive, engineering, shipbuilding, and other heavy industries of Glasgow were strongly affected. Furthermore, in the period under review there have been several changes in the categories of insurable female and juvenile workers, and the numbers of registered unemployed are therefore not comparable.

Lastly, unemployment among insured adult males relative to the entire ward population indicates to some extent the effects of unemployment upon families as a whole. It might have been desirable to examine the relationship between tuberculosis and economic status by using a positive index of employment rather than an index of unemployment. But unfortunately such investigation is impossible, since employment records are in no way related to the residence of the persons employed: the exchange of employment books is normally carried out in the area of the head office, or possibly of the accounting section, of the employers.

The unemployment index finally selected may, perhaps, in times of full or near-full employment in the city, be more representative of poverty than of the effects of unemployment itself; but in times of depression and crisis the proportions of adult males unemployed were highest not in the poorest wards but in those with skilled and industrial populations. The relationship between unemployment and a disease such as respiratory tuberculosis with an undetermined incubation period is inevitably difficult to investigate from past records: it would be desirable to relate tuberculosis incidence and mortality to previous, and particularly to long-term, unemployment. But this again was found impossible since records of the duration of unemployment by residence are not available.

*Poverty.*—The "valuation" index is based on the rents of houses in each ward and the percentage of houses with high rentals; it is calculated by relating the number of houses of over £45 rental to the numbers of houses of various lower rentals. This is taken as an inverse measure of the poverty of the ward: in fact, it measures, not poverty in an area, but the proportion of well-to-do houses in relation to various other categories and is therefore an index of "wealth" rather than of poverty. Its weakness is that it does not differentiate sufficiently between the poorer and the poorest wards, and does not distinguish greatly between stages of poverty. But it discriminates clearly between degrees of "wealth", and

* A "house" in the Scottish sense is a self-contained dwelling with its own entrance, and may be subdivided or shared between two or more households while still counting as a single house.

separates accurately the wealthy and moderately well-off wards from each other and from the poorer wards.

The poverty index used for the post-war period is a direct one, based on the number of persons in each ward in receipt of assistance from the Welfare Department of the Corporation of Glasgow, chargeable on the Ordinary and Able-bodied Outdoor Rolls, These comprised applicants for ordinary poor-law relief, together with their dependants, and able-bodied unemployed applicants who had exceeded the statutory period of receipt of unemployment insurance; but persons in institutions and persons receiving domiciliary assistance owing to blindness were excluded. The percentages of "ordinary and able-bodied poor" in the post-war years in relation to the total ward populations give a measure of poverty, as distinct from unemployment, in the wards. This index has not been used for any pre-war period, since the numbers chargeable on the Ordinary Outdoor Roll in the 1930s, during and after the long depression, were tremendously increased by the unemployed; hence for the 1930s the distribution of persons on relief is highly correlated with the unemployment distribution and does not give an independent measure of poverty.

The "relief" index has the opposite weakness to the "valuation" index, in that it does not differentiate greatly between the better-off and average wards, since in almost every area there are a few families dependent on relief. "Poor-law relief" indicates a degree of destitution rather beyond the ordinary meaning of poverty, and the index therefore does discriminate between the extreme stages of poverty, and does separate the very poorest wards from each other and from the better-off areas.

C.2. Sources of Data.—Through the co-operation and assistance of the Glasgow Public Health Department, the deaths and cases of respiratory and non-respiratory tuberculosis in each of the wards of Glasgow in the years 1930–47 were obtained. The annual births in each ward and the deaths of infants under one year old were published in the Reports of the Glasgow Public Health Department, as were also the annual deaths from all causes combined.

Estimates of ward populations and numbers of houses were for some years published in the Reports of the Glasgow Public Health Department; for other years, estimates were obtained with the aid of the Medical Officer of Health.

Overcrowding data were taken from the Report on the 14th Decennial Census of Scotland (1931) and from the Report on Overcrowding issued after the survey made under the Housing (Scotland) Act of 1935.

The rating of houses in each ward, from which valuation figures have been calculated, was also extracted from data in these reports.

Unemployment figures for the ten Employment Exchange Areas were obtained from the Statistics Division of the Ministry of Labour and National Service; these figures, covering large areas of Glasgow, were analysed and broken down for the smaller ward units with the assistance of the managers and staffs of the Glasgow Employment Exchanges.

The numbers of persons receiving Relief or Public Assistance, and chargeable on the Outdoor Rolls of the Welfare Department of the Corporation of Glasgow, were obtained from the Annual Reports published by that Department.

Tuberculosis deaths and notifications in Scotland as a whole and in the cities of Scotland since 1900 were published in the Annual Reports of the Registrar-General for Scotland and in the Public Health Reports of the various burghs.

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# PSYCHIATRIC ASPECTS OF SOMATIC IMMUNITY
## DIFFERENTIAL INCIDENCE OF PHYSICAL DISEASE IN THE HISTORIES OF PSYCHIATRIC PATIENTS

BY

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There have been few studies in the past of the incidence of physical disease in the mentally ill relative to that of the general population. Many writers have pointed to the concurrent or subsequent development of such disorders as pulmonary tuberculosis or enteric fever during the sojourn of psychiatric patients in mental hospitals, and there are a considerable number of reports from physicians concerning the intercurrence of mental symptoms in a variety of infections, traumata, tumours, intoxications, and other stressful experiences. This latter literature has been referred to in another communication (Lovett Doust, 1952), and an assessment of physical disorders present in patients admitted to a mental hospital has been made by Phillips (1937). In addition to these observations, a small number of studies have been concerned with the relative frequency of physical complaints in neurotically disturbed patients (Eysenck, 1947), with the length of convalescence of psychiatric patients afflicted by various bodily illnesses (Brodman and others, 1947a), and with the increased tendency of apparently mentally healthy patients hospitalized for physical illness to show neurotic scores on a psychological test (Cornell Service Index) as compared with other subjects who had not broken down (Brodman and others, 1947b). The current interest in the general adaptation syndrome of Selye (1950), as it affects the capacity for homeostatic adaptation of mentally-ill patients (Hoagland and Pincus, 1950) has been extended into the field of immunity, especially by the work of Dougherty and others (1945) on changing lymphocyte counts, these cells being thought to be concerned in immunity production (White and Dougherty, 1946).

It is clinically not infrequent that both the general physician and the psychiatrist have the opportunity of witnessing what may be called the alternation of psychiatric and somatic disease in the course of the illness of an individual patient. This alternation may take the form, for example, of a confusional episode associated with pneumonia and occur before, during the height of, or after the pyrexia; it may be observed in the form of a psychotic reaction, similar in symptomatology and appearance to an "idiopathic" psychiatric syndrome, perhaps as the sequela of an attack of influenza or of an apparently normal pregnancy and labour; or it may occur as a somatic illness during the investigation and treatment of a mental disturbance and present as bronchial asthma, rheumatoid arthritis, etc. (Appel and Rosen, 1950).

The present paper stems from the clinical impression in the wards of a psychiatric

hospital that the somatic illness history loading of the patients seen there appeared heavier than that of a control group of healthy subjects. This impression led to an endeavour to assess the known factors concerned in the development of immunity responses and it was surprising to the writer that these factors are both so few and so relatively unimportant. Apart from the dose of the infecting organism, most of them seem to be accounted for by age, sex, race, diet, state of nutrition, climate, and seasonal variation, together with a group of "X" factors relating to individual susceptibility and resistance about which little is known. It is of some psychiatric interest that one of these "X" factors may be that of fatigue, since it has been shown that susceptibility is increased when fatigue is present (Boycott and Price-Jones, 1926). The evidence however suggests that fatigue tended to act as a factor reactivating existing infection rather than as one predisposing to new infection. Perhaps the most obvious of the known factors listed above is state of nutrition, yet even here (as, for example, the investigations of Gell (1948) and of Balch (1950) have shown), it requires the most extreme forms of under-nutrition to effect a significant change in the ability to form antibodies against inoculated antigens and only pre-mortal states of under-nourishment can materially alter the individual threshold. Various components of the diet have been implicated but none has been shown to be of general importance; indeed the evidence for indicting a number of vitamins appears to be both confusing and extremely flimsy. As Wilson and Miles (1950) point out, the vitamin deficiencies which seem to have some definite effect in this respect are avitaminoses A and C. Of these the former appears to be of major importance only during the period of rapid bodily growth, while both deficiencies seem to influence resistance only when the lack of the factor is a gross one. Furthermore, apart from Gell's work, most investigations have been done on animals, in whom the whole pattern of individual response to human infections is so often different from that of man.

In recent years evidence has begun to accumulate in a rather different direction towards the elucidation of certain hitherto neglected factors concerned in the predisposition to infective and other disease processes. An orientation of medicine, loosely called the "psychosomatic" approach, based on a growing understanding of psychophysical relationships in man, has come into being. The integration of emotional factors with those of the somatic constitution has repercussions, both in the management of the individual patient and in the understanding of the immunological implications of why a particular individual fell sick with a particular malady at a particular time. This aspect of immunology is considered by Halliday (1948); it may be exemplified for upper respiratory infection by the recent monograph produced under the direction of H. G. Wolff of the New York Hospital (Holmes and others, 1950).

The present paper reports a study designed to show that personality factors play a part at least as important as other better-recognized factors in determining the resistance of individuals to various disease processes. It is the first limb of an investigation presenting a point of view in immunology which has been largely neglected in the past but may prove of fundamental importance to an understanding of disease

as a reaction of the total organism to stresses requiring to be met by homeostatic adaptation, even though such an adaptation may be harmful and even fatal to the individual making it.

### Experimental Method

A list of 110 disorders was compiled, 38 of which represented symptoms and complaints of a bodily nature and the remaining 72 actual somatic diseases and syndromes. The selection was made arbitrarily, those illnesses being included which were thought to represent a significant incidence in the population at risk. The disorders investigated are listed in Table I (pp. 52 to 57) where they are grouped by systems affected. Medical terminology was kept to a minimum, especially in the case of complaints, preference being given to popular lay terms which could be presumed to be understood by the majority of the subjects investigated. Fashions in the designation of aches and pains change with the times, and there is a considerable difference in local geography in this respect, different parts of the country having their own terms for certain disorders, but most of the terms used were readily comprehended by the subjects studied.

The list was prepared in questionnaire form, and instructions were added that the subject was to underline all those symptoms and illnesses from which he had ever suffered. He was assured that his answers would be treated confidentially, that his record would be used as in a census to represent an individual tally with respect to a total, and that no personal considerations would accrue from the pattern of his responses.

It was recognized at the outset that an investigation such as this could only approach validity providing sufficient healthy control subjects were used to give the results country-wide significance. The sampling methods employed and the techniques used in estimating reliability will be discussed later in this paper; let it suffice to say here that when copies of the questionnaire were filled in by the investigator after questioning a small batch of controls and were compared with those returned by mail from different parts of the country, no significant difference was found either in the absolute numbers of disorders recorded or in their differential pattern.

A consideration of the list of disorders will reveal no specifically gynaecological complaints or illnesses. For reasons dependent upon the sampling method employed, fewer females than males were available for study, and it was deemed advisable therefore to limit the disorders to those in which sex differences were not weighted in favour of female morbidity. In point of fact, little difference was found between the sexes for the disorders studied and for this reason no breakdown of the figures by sex is given.

### Method of Obtaining Data

Apart from the small number of controls personally interviewed as already mentioned, the remaining healthy subjects were simply requested to fill out the questionnaire. This simple procedure did not, however, apply to most of the psychiatric patients. The neurotic group and those with character disorders were usually able to complete the questionnaire unaided, but for the psychotic groups personal interviews were used, each disorder being taken separately and the patient being asked if he had ever suffered from the disease or complaint in question. These answers, together with those of the other psychiatric groups included, were then checked by physical examination, interrogation of relatives, the medical history report of the hospital, and the records of other hospitals to which the patient had formerly been admitted. A final evaluation depended upon the sum of all the information garnered from these various sources. Surprisingly little extra information was however actually added by these time-consuming measures; except in the case of grossly deteriorated psychotics, the patients' self-assessments agreed well with the evidence supplied through such alternative channels.

TABLE I

PERCENTAGE INCIDENCE OF 38 BODILY COMPLAINTS AND 72 ORGANIC DISORDERS

| Tissue or Organ System, etc., Involved | | Complaint or Disorder | Percentage Incidence of Somatic | | | | |
|---|---|---|---|---|---|---|---|
| | | | | | Psycho-Neurosis | | |
| | | | Controls (354) | Psycho-paths (42) | Anxiety States (61) | Hysterics (59) | Total (120) |
| Infectious Fevers of Childhood | | Measles | 74·9 | 59·5 | 82·0 | 59·3 | 70·8 |
| | | Rubella | 12·4 | 35·0 | 23·5 | 11·1 | 19·2 |
| | | Mumps | 43·2 | 47·6 | 47·5 | 28·8 | 38·3 |
| | | Chickenpox | 41·5 | 38·1 | 49·2 | 39·0 | 44·2 |
| | | Scarlatina | 21·5 | 35·7 | 27·9 | 22·0 | 25·0 |
| | | Diphtheria | 7·1 | 15·0 | 11·8 | 11·1 | 11·5 |
| | | Pertussis | 30·5 | 33·3 | 31·1 | 28·8 | 30·0 |
| | | Mastoiditis | 2·0 | 5·0 | 11·8 | 0 | 7·7 |
| | | Rheumatic fever | 1·7 | 2·4 | 13·1 | 10·2 | 11·7 |
| Congenital Defects, etc. | | Flat feet | 8·9 | 4·5 | 9·1 | 12·0 | 10·6 |
| | | Fallen arches | 4·0 | 0 | 4·5 | 4·0 | 4·3 |
| | | Hernia | 7·4 | 4·5 | 6·8 | 4·0 | 5·3 |
| | | Undescended testis | 1·5 | 4·5 | 6·8 | 6·0 | 5·4 |
| | | Impacted wisdom teeth | 0·5 | 0 | 6·8 | 6·0 | 5·4 |
| | | Varicose veins | 4·0 | 9·1 | 4·5 | 4·0 | 4·3 |
| | | Infantile fits | 1·5 | 0 | 9·1 | 2·0 | 5·4 |
| Other Childhood Complaints and Disorders | | Sore throat | 70·4 | 75·0 | 82·4 | 100·0 | 88·5 |
| | | Tonsillectomy | 30·3 | 50·0 | 29·4 | 33·3 | 30·8 |
| | | Ear disease | 6·6 | 20·0 | 47·1 | 0 | 30·8 |
| | | Meningitis | 1·4 | 0 | 6·6 | 1·6 | 4·2 |
| | | Worm infestation | 4·0 | 9·1 | 4·5 | 2·0 | 3·2 |
| | | Rickets | 0·6 | 0 | 4·9 | 0 | 2·5 |
| | | Scurvy | 0 | 0 | 2·3 | 0 | 1·1 |
| Respiratory System | Complaints | Shortness of breath | 12·4 | 18·2 | 36·4 | 32·0 | 34·0 |
| | | Catarrh | 13·8 | 21·4 | 27·9 | 13·6 | 20·8 |
| | | Persistent cough | 8·8 | 26·2 | 19·7 | 13·6 | 16·7 |
| | | Haemoptysis | 0·7 | 0 | 5·9 | 0 | 3·8 |
| | Disorders | Frequent colds | 52·3 | 71·4 | 55·7 | 55·9 | 55·8 |
| | | Feverish cold | 14·5 | 20·0 | 23·5 | 33·3 | 26·9 |
| | | Laryngitis | 11·2 | 10·0 | 17·6 | 11·1 | 15·4 |
| | | Bronchitis | 8·2 | 19·0 | 19·7 | 20·3 | 20·0 |
| | | Influenza | 52·5 | 38·1 | 67·2 | 57·6 | 62·5 |
| | | Pleurisy | 2·5 | 5·0 | 17·6 | 11·1 | 15·4 |
| | | Pneumonia | 10·7 | 14·3 | 18·0 | 10·2 | 14·2 |
| | | Tuberculosis | 1·9 | 2·4 | 1·6 | 1·7 | 1·7 |
| | | Bronchiectasis | 0·3 | 2·4 | 0 | 1·7 | 0·8 |
| Allergy | | Asthma | 1·7 | 0 | 6·6 | 1·7 | 4·2 |
| | | Hay fever | 4·8 | 7·1 | 9·8 | 10·2 | 10·0 |
| | | Eczema | 11·0 | 26·2 | 18·0 | 5·1 | 11·7 |
| | | Urticaria | 3·1 | 2·4 | 6·6 | 8·5 | 7·5 |
| | | Sensitive skin | 5·3 | 10·0 | 11·8 | 11·1 | 11·5 |

The degree of significance was computed from $\chi^2$ tables in which the contributions of each of the psychiatric diagnostic groups (controls, psychopaths, neurotics, depressives and schizophrenics) could be assessed separately with respect to the total $\chi^2$ value (see Table II for example).

| Complaint or Disorder | | | $\chi^2$ | Confidence Level of Probability *P* Per cent. | Principal Psychiatric Disorder contributing to this $\chi^2$ | Extent of Contribution to Total $\chi^2$ |
|---|---|---|---|---|---|---|
| Psychosis | | | | | | |
| Depressives (59) | Schizophrenics (51) | Total (110) | | | | |
| 79·7 | 86·3 | 82·7 | 4·6 | N.S. | — | — |
| 28·8 | 29·4 | 29·1 | 21·1 | 0·1 | All | — |
| 44·1 | 35·3 | 40·0 | 1·2 | N.S. | — | — |
| 54·2 | 52·9 | 53·6 | 1·9 | N.S. | — | — |
| 22·0 | 27·5 | 24·5 | 4·6 | N.S. | — | — |
| 16·9 | 19·6 | 18·2 | 12·6 | 2·5 | Psychosis | 8·4 |
| 55·9 | 56·9 | 56·4 | 17·7 | 0·5 | Psychosis | 14·8 |
| 3·4 | 2·0 | 2·7 | 3·0 | N.S. | — | — |
| 6·8 | 7·8 | 7·3 | 22·0 | 0·1 | Neurosis | 13·1 |
| — | — | — | 0·8 | N.S. | — | — |
| — | — | — | 0·02 | N.S. | — | — |
| — | — | — | 0·6 | N.S. | — | — |
| — | — | — | 5·2 | N.S. | — | — |
| — | — | — | 10·1 | 1·0 | Neurosis | 7·0 |
| — | — | — | 1·2 | N.S. | — | — |
| — | — | — | 4·6 | 5·0 | Neurosis | 2·8 |
| 91·5 | 96·1 | 93·6 | 22·0 | 0·1 | Schizophrenia | 7·7 |
| 35·6 | 37·3 | 36·4 | 3·6 | N.S. | — | — |
| 13·6 | 23·5 | 18·2 | 17·8 | 0·5 | Neurosis | 6·5 |
| 3·4 | 0 | 1·8 | 7·3 | N.S. | — | — |
| — | — | — | 1·6 | N.S. | — | — |
| 6·8 | 5·9 | 6·4 | 16·0 | 0·5 | Depression | 7·4 |
| — | — | — | — | N.S. | — | — |
| — | — | — | 15·8 | 0·1 | Neurosis | 11·0 |
| 39·0 | 31·4 | 35·5 | 21·3 | 0·1 | Depression | 11·6 |
| 25·4 | 29·4 | 27·3 | 23·6 | 0·1 | Schizophrenia | 7·7 |
| 3·4 | 9·8 | 6·4 | 11·7 | 2·5 | Schizophrenia | 8·4 |
| 71·2 | 78·5 | 74·5 | 8·6 | N.S. | — | — |
| 28·8 | 54·9 | 40·9 | 33·8 | 0·1 | Schizophrenia | 23·7 |
| 20·3 | 9·8 | 15·5 | 4·0 | N.S. | — | — |
| 15·3 | 19·6 | 17·3 | 14·2 | 1·0 | Neurosis | 5·0 |
| 98·3 | 80·4 | 90·0 | 40·9 | 0·1 | Depression | 14·4 |
| 11·9 | 9·8 | 10·9 | — | — | — | — |
| 18·6 | 21·6 | 20·0 | 5·8 | N.S. | — | — |
| 6·8 | 3·9 | 5·5 | 5·4 | N.S. | — | — |
| 0 | 2·0 | 0·9 | 4·9 | N.S. | — | — |
| 1·7 | 3·9 | 2·7 | 4·1 | N.S. | — | — |
| 13·6 | 19·6 | 16·4 | 13·6 | 1·0 | Schizophrenia | 6·1 |
| 50·8 | 45·1 | 48·2 | 67·7 | 0·1 | Depression | 33·2 |
| 10·2 | 11·8 | 10·9 | 11·8 | 2·5 | Schizophrenia | 4·2 |
| 27·1 | 33·3 | 30·0 | 27·6 | 0·1 | Schizophrenia | 11·7 |

TABLE I—*cont.*

| Tissue or Organ System, etc., Involved | | Complaint or Disorder | Percentage Incidence of Somatic | | | | |
|---|---|---|---|---|---|---|---|
| | | | | | Psycho-Neurosis | | |
| | | | Controls (354) | Psycho-paths (42) | Anxiety States (61) | Hysterics (59) | Total (12 |
| Gastro-Intestinal Tract | Complaints | Constipation | 20·6 | 23·8 | 41·0 | 27·1 | 32· |
| | | Diarrhoea | 29·1 | 23·8 | 44·3 | 18·6 | 31· |
| | | Stomach troubles | 7·6 | 14·3 | 24·6 | 22·0 | 23· |
| | | Indigestion | 20·4 | 20·0 | 52·9 | 77·8 | 61· |
| | | Gastric stomach | 2·6 | 5·0 | 0 | 22·2 | 7· |
| | | Pain after meals | 3·9 | 10·0 | 41·2 | 22·2 | 34· |
| | | Nausea and vomiting | 17·8 | 30·0 | 52·9 | 55·6 | 53· |
| | | Bilious attacks | 13·6 | 19·0 | 27·9 | 27·1 | 27· |
| | | Vomiting blood | 0·7 | 0 | 5·9 | 0 | 3· |
| | | Blood in stools | 0 | 5·0 | 11·8 | 22·2 | 15· |
| | | Colic | 4·6 | 0 | 5·9 | 0 | 3· |
| | | Jaundice | 5·9 | 0 | 17·6 | 11·1 | 15· |
| | Disorders | Appendicitis | 9·3 | 10·0 | 11·8 | 11·1 | 11· |
| | | Chronic appendix | 3·9 | 45·0 | 24·4 | 33·3 | 30· |
| | | Appendectomy | 13·8 | 20·0 | 5·9 | 11·1 | 7· |
| | | Peritonitis | 1·3 | 5·0 | 0 | 0 | 0 |
| | | Peptic ulcer | 1·7 | 0 | 2·3 | 0 | 0· |
| | | Piles | 9·0 | 9·5 | 16·4 | 1·7 | 9· |
| | | Dysentery | 5·4 | 25·0 | 11·8 | 11·1 | 11· |
| | | Gallstones | 0·3 | 0 | 0 | 0 | 0 |
| | | Infective hepatitis | 2·6 | 0 | 5·9 | 0 | 3· |
| Central Nervous System | Complaints | Headache | 32·8 | 31·0 | 47·5 | 35·6 | 41· |
| | | Recurrent headache | 7·2 | 25·0 | 35·3 | 55·6 | 42· |
| | | Vertigo | 5·3 | 35·0 | 41·2 | 33·3 | 38· |
| | | Blurred vision | 7·2 | 30·0 | 35·3 | 22·2 | 30· |
| | | Syncope | 12·4 | 38·1 | 42·6 | 59·3 | 50· |
| | | "Turns" | 0·8 | 20·0 | 17·6 | 0 | 11· |
| | | "Absences" | 1·5 | 0 | 4·5 | 0 | 2· |
| | | *Déjà vu* | 10·4 | 22·7 | 22·7 | 14·0 | 18· |
| | | Depersonalization | 0·5 | 4·5 | 2·3 | 2·0 | 2· |
| | | "Watched self act" | 0·5 | 0 | 2·3 | 0 | 1· |
| | | Collapse | 0 | 0 | 9·1 | 12·0 | 10· |
| | Disorders | Head injury | 24·3 | 27·5 | 54·5 | 40·0 | 46· |
| | | Accident | 23·3 | 27·3 | 25·0 | 40·0 | 33· |
| | | Heat stroke | 1·3 | 5·0 | 17·6 | 11·1 | 15· |
| | | Herpes zoster | 0·8 | 0 | 1·6 | 1·7 | 1· |
| | | Neuritis | 7·6 | 7·1 | 26·2 | 15·2 | 20· |
| | | Migraine | 8·2 | 11·9 | 37·7 | 25·4 | 31· |
| | | Fits (epilepsy) | 0·8 | 4·8 | 4·9 | 6·8 | 5· |
| Cardiovascular System | Complaints | Palpitation | 5·4 | 0 | 13·6 | 14·0 | 13· |
| | | Epistaxis | 21·1 | 25·0 | 52·9 | 22·2 | 42· |
| | | Swollen ankles | 3·5 | 4·5 | 6·9 | 2·0 | 4· |
| | Disorders | Heart trouble | 1·1 | 2·4 | 4·9 | 3·4 | 4· |
| | | Blood pressure | 1·1 | 0 | 4·9 | 5·1 | 5· |
| | | Anaemia | 2·3 | 4·8 | 1·6 | 3·4 | 2· |

| Complaint or Disorder | | | $\chi^2$ | Confidence Level of Probability *P* Per cent. | Principal Psychiatric Disorder contributing to this $\chi^2$ | Extent of Contribution to Total $\chi^2$ |
|---|---|---|---|---|---|---|
| Psychosis | | | | | | |
| Depressives (59) | Schizophrenics (51) | Total (110) | | | | |
| 79·7 | 74·5 | 77·3 | 85·9 | 0·1 | Depression | 41·4 |
| 54·2 | 62·7 | 58·2 | 23·5 | 0·1 | Psychosis | 19·1 |
| 49·1 | 37·3 | 43·6 | 67·8 | 0·1 | Depression | 34·2 |
| 74·6 | 68·6 | 71·8 | 49·8 | 0·1 | Psychosis | 28·0 |
| 30·5 | 11·8 | 21·8 | 27·7 | 0·1 | Depression | 12·1 |
| 37·3 | 51·0 | 43·6 | 53·5 | 0·1 | Psychosis | 28·9 |
| 66·1 | 74·5 | 70·0 | 46·5 | 0·1 | Psychosis | 24·0 |
| 39·0 | 66·7 | 41·8 | 63·1 | 0·1 | Schizophrenia | 41·2 |
| 0 | 5·9 | 2·7 | 8·7 | N.S. | — | — |
| 6·8 | 17·6 | 11·8 | 24·1 | 0·1 | Schizophrenia | 15·7 |
| 15·3 | 9·8 | 12·7 | 9·2 | N.S. | — | — |
| 8·5 | 13·7 | 10·9 | 6·4 | N.S. | — | — |
| 13·6 | 13·7 | 13·6 | 3·5 | N.S. | — | — |
| 44·1 | 35·3 | 40·0 | 58·7 | 0·1 | Depression | 17·4 |
| 10·2 | 13·7 | 11·8 | 1·7 | N.S. | — | — |
| 1·7 | 3·9 | 2·7 | 2·9 | N.S. | — | — |
| 8·5 | 1·9 | 5·5 | 13·5 | 1·0 | Depression | 12·4 |
| 39·0 | 23·5 | 31·8 | 39·9 | 0·1 | Depression | 30·6 |
| 18·6 | 9·8 | 14·5 | 21·0 | 0·1 | Depression | 8·2 |
| 3·4 | 0 | 1·8 | — | N.S. | — | — |
| 5·1 | 3·9 | 4·5 | 1·6 | N.S. | — | — |
| 74·6 | 96·1 | 84·5 | 55·8 | 0·1 | Schizophrenia | 32·6 |
| 55·9 | 43·1 | 50·0 | 63·8 | 0·1 | Depression | 26·1 |
| 39·0 | 49·0 | 43·6 | 45·5 | 0·1 | Schizophrenia | 13·8 |
| 40·7 | 43·1 | 41·8 | 34·5 | 0·1 | Psychosis | 17·0 |
| 40·7 | 54·9 | 47·3 | 75·1 | 0·1 | Neurosis | 26·3 |
| 15·3 | 31·4 | 22·7 | 41·4 | 0·1 | Schizophrenia | 22·8 |
| — | — | — | — | N.S. | — | — |
| — | — | — | 13·4 | 0·5 | Neurosis | 8·1 |
| — | — | — | 3·6 | N.S. | — | — |
| — | — | — | 0·5 | N.S. | — | — |
| — | — | — | — | N.S. | — | — |
| — | — | — | 10·7 | 0·5 | Neurosis | 7·6 |
| — | — | — | 2·5 | N.S. | — | — |
| 15·3 | 9·8 | 12·7 | — | — | — | — |
| 10·2 | 5·9 | 8·2 | 25·5 | 0·1 | Depression | 18·5 |
| 44·1 | 43·1 | 43·6 | 70·0 | 0·1 | Psychosis | 50·0 |
| 22·0 | 13·7 | 18·2 | 35·8 | 0·1 | Neurosis | 23·2 |
| 3·4 | 15·6 | 9·1 | 30·1 | 0·1 | Schizophrenia | 21·0 |
| — | — | — | 7·8 | 2·5 | Neurosis | 5·0 |
| 28·8 | 62·7 | 44·5 | 22·6 | 0·1 | Schizophrenia | 16·0 |
| — | — | — | 1·5 | N.S. | — | — |
| 3·4 | 9·8 | 6·4 | 13·8 | 1·0 | Schizophrenia | 9·5 |
| 13·6 | 19·6 | 16·4 | 46·4 | 0·1 | Schizophrenia | 24·6 |
| 22·0 | 25·5 | 23·6 | 66·0 | 0·1 | Schizophrenia | 30·6 |

TABLE I—*cont.*

| Tissue or Organ System, etc., Involved | | Complaint or Disorder | Percentage Incidence of Somatic | | | | |
|---|---|---|---|---|---|---|---|
| | | | | | Psycho-Neurosis | | |
| | | | Controls (354) | Psycho-paths (42) | Anxiety States (61) | Hysterics (59) | Total (120) |
| Locomotor System | Complaints | Muscle strain | 7·9 | 20·0 | 35·3 | 33·3 | 34·6 |
| | | Back pain | 11·6 | 23·8 | 31·4 | 23·7 | 27·5 |
| | | Neck pain | 6·6 | 15·0 | 35·3 | 22·2 | 30·8 |
| | | Joint pain | 1·5 | 4·5 | 4·5 | 16·0 | 10·6 |
| | | "Writer's cramp" | 3·3 | 20·0 | 5·9 | 33·3 | 15·4 |
| | Disorders | Lumbago | 2·0 | 0 | 4·5 | 2·0 | 3·2 |
| | | Sciatica | ·05 | 0 | 4·5 | 0 | 2·1 |
| | | Muscular rheumatism | 12·1 | 21·4 | 18·0 | 10·2 | 14·2 |
| | | Fibrositis | 3·0 | 0 | 2·3 | 2·0 | 2·1 |
| | | Arthritis | 4·5 | 11·9 | 3·3 | 5·1 | 4·2 |
| Skin | Complaints | Skin eruptions | 6·4 | 9·9 | 13·1 | 10·2 | 11·7 |
| | | Itchy skin | 6·8 | 9·5 | 18·0 | 10·2 | 14·2 |
| | | Anal pruritus | 5·9 | 4·5 | 6·8 | 8·0 | 7·4 |
| | Disorders | Dermatitis | 12·7 | 28·6 | 24·6 | 6·8 | 15·8 |
| | | Acne | 7·1 | 9·5 | 13·1 | 0 | 6·7 |
| | | Impetigo | 14·9 | 13·6 | 4·5 | 4·0 | 4·3 |
| | | Scabies | 15·8 | 14·3 | 19·7 | 3·4 | 11·7 |
| | | Boils | 33·6 | 45·0 | 76·5 | 22·2 | 57·7 |
| Genito-Urinary Tract Disorders | | Kidney and Bladder | 6·5 | 7·1 | 11·5 | 15·3 | 13·3 |
| | | Gonorrhoea | 3·0 | 9·1 | 1·6 | 0 | 1·1 |
| | | Syphilis | 2·5 | 0 | 0 | 0 | 0 |
| Miscellaneous | | Fracture | 19·5 | 21·4 | 21·3 | 22·0 | 21·7 |
| | | Operation | 12·4 | 21·4 | 16·4 | 11·9 | 14·2 |
| | | Thyroid and Goitre | 1·7 | 0 | 1·6 | 1·7 | 1·7 |

## Population Sampling and Clinical Material

A total of 626 subjects was investigated, comprising 354 controls, healthy at the time of examination, and 272 psychiatric patients in whom no gross physical disability was apparent clinically.

Psychiatric Patients.—This group included 120 cases of psychoneurosis (61 anxiety states and 59 hysterics), 42 cases of psychopathic personality, 59 cases of affective disorder (all in a depressive phase of their illness), and 51 cases of schizophrenia. Patients in whom reasonable doubt existed about the diagnosis or in whom double diagnoses had been made (*e.g.* "schizo-affective disorder") were excluded from the investigation, as were cases of probable organic neurological abnormality and patients in whom a degree of mental defect was complicating the clinical picture. Within these limits, however, were included most of the major categories of mental disorder, ranging from acute anxiety states present for only a few months, up to psychoses which had curbed the patient's socialization for 20 years.

All the psychotic patients, twenty of the psychopaths, and 26 of the neurotics, were

| Complaint or Disorder | | | $\chi^2$ | Confidence Level of Probability *P* Per cent. | Principal Psychiatric Disorder contributing to this $\chi^2$ | Extent of Contribution to Total $\chi^2$ |
|---|---|---|---|---|---|---|
| Psychosis | | | | | | |
| Depressives (59) | Schizophrenics (51) | Total (110) | | | | |
| 15·3 | 39·2 | 26·4 | 29·2 | 0·1 | Schizophrenia | 18·2 |
| 42·4 | 52·9 | 47·3 | 53·2 | 0·1 | Schizophrenia | 22·9 |
| 20·3 | 45·1 | 31·8 | 24·2 | 0·1 | Schizophrenia | 20·5 |
| — | — | — | 12·2 | 0·5 | Neurosis | 8·3 |
| 6·8 | 31·4 | 18·2 | 30·6 | 0·1 | Schizophrenia | 20·3 |
| — | — | — | 0·9 | N.S. | — | — |
| — | — | — | 2·2 | N.S. | — | — |
| 35·6 | 56·9 | 45·5 | 57·4 | 0·1 | Schizophrenia | 38·5 |
| — | — | — | 0·8 | N.S. | — | — |
| 27·1 | 23·5 | 25·5 | 46·5 | 0·1 | Psychosis | 36·8 |
| 8·5 | 17·6 | 12·7 | 25·2 | 0·1 | Schizophrenia | 11·1 |
| 33·9 | 35·3 | 34·5 | 49·6 | 0·1 | Psychosis | 38·0 |
| — | — | — | 0·3 | N.S. | — | — |
| 50·8 | 45·1 | 48·2 | 54·7 | 0·1 | Depression | 26·2 |
| 28·8 | 23·5 | 26·4 | 15·6 | 0·5 | Schizophrenia | 8·2 |
| — | — | — | 6·9 | 5·0 | Controls | 4·3 |
| 15·3 | 21·6 | 18·2 | 2·4 | N.S. | — | — |
| 54·2 | 64·7 | 59·1 | 10·8 | 5·0 | Schizophrenia | 4·2 |
| 23·7 | 31·4 | 27·3 | 33·9 | 0·1 | Schizophrenia | 17·5 |
| 0 | 0 | 0 | 4·1 | N.S. | — | — |
| 0 | 0 | 0 | — | N.S. | — | — |
| 23·7 | 27·5 | 25·5 | 1·7 | N.S. | — | — |
| 18·6 | 33·3 | 25·5 | 13·9 | 1·0 | Schizophrenia | 10·3 |
| 1·7 | 3·9 | 2·7 | 2·9 | N.S. | — | — |

drawn from the wards and out-patient clinics of the Maudsley Hospital, and from the observation ward of St. Francis Hospital, Dulwich.

The remaining 116 patients were Army personnel who had broken down psychiatrically after their induction into the Service. All had been seen previously by more than one Army psychiatrist and all had been deemed sufficiently sick to be recommended for discharge on psychiatric grounds. All members of this military group were British "other ranks"; they comprised fifty anxiety states, 44 hysterics, and 22 cases of psychopathic personality.

The military patients were all males (age range: 18–46, mean 22·8 years). The hospital patients included 37 female and 22 male depressives (age range: 19–75, mean 36·4 years), 22 female and 29 male schizophrenics (age range: 15–46, mean 27·1 years), four female and sixteen male cases of psychopathic personality (age range: 18–34, mean 22·1 years), and ten female and sixteen male neurotics (age range: 14–60, mean 31·1 years).

CONTROL GROUP.—The healthy controls represented a similar dichotomy of sampling. 152 of them were civilians: physicians, nurses, hospital workers, business men, and housewives, and the remaining 202 were healthy soldiers drawn from a neighbouring R.A. depot.

The military group included recent inductees from all over the country, troops who had lately returned from overseas, and regular soldiers awaiting drafting to other units. The investigation was carried out in 1948–49, *i.e.* during the period of conscription but after the end of the war. The bias of self-selection was avoided since the soldiers did not volunteer for this investigation, but were detailed to report to the Institute of Psychiatry by special arrangement with the War Office.

All members of the military control groups were males (age range: 18–52, mean 25·5 years). Of the civilians, 63 were females and 89 males (age range: 16–68, mean 31·2 years).

STATE OF HEALTH.—Since the investigation concerned the differential incidence of somatic disorders experienced by the subjects in the *past*, every effort was made to exclude subjects suffering from any of these disorders. Both the psychiatric and the control groups were free from any ascertainable bodily *disease* at the time of examination, though of course the same could not be said of their *symptoms*, for psychiatric patients are known to experience complaints referable to the soma, and such symptoms are not readily checked by reference either to the experience of relatives or to the medical record. It was therefore thought desirable sharply to distinguish bodily complaints from bodily diseases in the analysis of the results and to express the figures for each group separately. Even so it may be mentioned here that the incidence of unjustifiable and hypochondriacal exaggeration of symptoms appeared to be low in all patient groups by comparison with their previous history of actual illnesses.

## RESULTS

These may be studied in detail in Table I. The statistical technique employed obeyed the strictures of Lewis and Burke (1949) concerning the $\chi^2$ method, in that the total expected incidence (actual incidence/total number) exceeded 0·24 for significance evaluation, and the differential contributory expected number had to be at least 10. The modified $\chi^2$ analysis used (exemplified in detail in Table II) enabled the actual contribution to the total $\chi^2$ to be computed for each diagnostic group and sub-group,

TABLE II

EXAMPLE OF METHOD OF COMPUTING DIFFERENTIAL $\chi^2$ CONTRIBUTION OF EACH DIAGNOSTIC GROUP

| Diagnostic Group | Influenza | | Expected Number | | Deviation from expected | | Contribution to $\chi^2$ | | |
|---|---|---|---|---|---|---|---|---|---|
| | Actual Incidence | Non-Incidence | Incidence | Non-Incidence | Incidence | Non-Incidence | Incidence | Non-Incidence | Total |
| Controls (354) | 186 | 168 | 212·75 | 141·25 | −26·75 | +26·75 | 3·37 | 5·07 | 8·44 |
| Psychopaths (42) | 16 | 26 | 25·24 | 16·76 | − 9·24 | − 9·24 | 3·38 | 5·09 | 8·47 |
| Neurotics (120) | 75 | 45 | 72·12 | 47·88 | + 2·88 | − 2·88 | 0·12 | 0·18 | 0·30 |
| Depressives (59) | 58 | 1 | 35·46 | 23·54 | +22·54 | −22·54 | 14·32 | 21·58 | 35·90 |
| Schizophrenics (51) | 41 | 10 | 30·65 | 20·35 | +10·35 | −10·35 | 3·48 | 5·26 | 8·74 |
| Total Subjects (626) | 376 | 250 | 376·22 | 249·78 | 0 | 0 | 24·67 | 37·18 | 61·85 |

Expected Incidence $=\frac{376}{626}=0{\cdot}601$; Expected Non-Incidence $=\frac{250}{626}=0{\cdot}399$; Total $\chi^2=61{\cdot}85$; $P<0{\cdot}001$

The outstanding history of influenza among the *depressive* population is clearly illustrated. Note that the contributions of the control and psychopath groups are negative and that the criteria of Lewis and Burke (1949) are fulfilled.

and so indicated which particular group, if any, was outstanding in having a history of the disorder under investigation. In this connection it will be noted (Table II) that the deviations from the expected incidence may be either positive or negative, since it is just as significant that, for example, controls and psychopaths do *not* tend to have influenza in their medical histories, as it is that cases of depression *do* have such a history.

In general it may be seen from Table I that there is a higher incidence of physical illness and of complaints referable to the body in the medical histories of psychiatric patients than there is in a comparable sample of the general population. From the analysis of diagnostic group differential incidences, it can also be seen that this increased history loading is heavier in the case of the psychotics than it is in that of the neurotics or the character disorders. But over and above these gross differences (seen additionally in Table III in which total illness loadings are analysed by diagnostic

TABLE III

ANALYSIS BY PSYCHIATRIC DIAGNOSIS OF TOTAL SOMATIC ILLNESS HISTORY LOADINGS

| Psychiatric Diagnosis | Total Somatic Complaints | | Total Somatic Diseases | |
|---|---|---|---|---|
| | Mean | S.D. | Mean | S.D. |
| Controls .. .. | 1·706 | 1·391 | 6·291 | 1·722 |
| Psychopaths .. .. | 2·201 | 3·674 | 6·800 | 4·845 |
| Neurotics .. .. | 3·491 | 6·532 | 8·907 | 2·799 |
| Depressives .. .. | 8·334 | 2·176 | 13·654 | 4·447 |
| Schizophrenics .. | 10·320 | 3·005 | 13·820 | 5·402 |

Note that the number of both complaints and diseases increases with severity of the mental disorder.

group), there is the interesting revelation that certain groups of disorders occur in the histories of certain psychiatric diagnostic groups more often than in others. Thus, for example, depressed patients, prior to their psychiatric disturbance, would seem to have suffered from disorders referable to the gastro-intestinal tract (chronic appendicitis, peptic ulcer, haemorrhoids, dysentery, and "stomach troubles") not only more than controls but also more than neurotics or schizophrenics, while schizophrenics similarly have a higher somatic illness experience of disorders referable to the locomotor and cardiovascular systems (hypertension, muscular rheumatism, "back and neck pains") than any of the other diagnostic groups. It is, in fact, possible to draw up tables based on the figures from which Table I is derived, to show the *positive* correlations between differential psychiatric diagnosis and the history of specific bodily disorders in the past (Table IV, overleaf), and between such psychiatric states and the *negative* incidence of other bodily disorders (Table V, overleaf). Thus, for example, it can be seen that whereas a history of pertussis was found significantly in the medical illness loading of patients who subsequently developed both depressive and schizophrenic psychoses, this disease has a negative correlation with the later development of neuroticism. These tables extend the more limited yet similar findings of Ross and others (1950).

TABLE IV

OUTSTANDING POSITIVE CORRELATIONS BETWEEN PRESENT MENTAL STATE AND DIFFERENTIAL PATTERN OF SPECIFIC COMPLAINTS AND DISEASES IN HISTORIES OF THREE TYPES OF PSYCHIATRIC DISORDER AS COMPARED WITH MEDICAL HISTORIES OF THE CONTROL GROUP

No particular positive correlations existed for the psychopathic group

| Psycho-Neurosis | | Depression | | Schizophrenia | |
|---|---|---|---|---|---|
| Symptoms | Diseases | Symptoms | Diseases | Symptoms | Diseases |
| *Déjà vu* | Rheumatic fever | Stomach trouble | Rickets | Headache | Whooping cough |
| Palpitations | Head injury | Constipation | Influenza | Bilious attacks | Bronchitis |
| Dyspnoea | Migraine | Indigestion | Eczema | Diarrhoea | Boils |
| | Syncope | Gastric stomach | Peptic ulcer | Nausea | Fits |
| | Impacted molars | Nasal catarrh | Herpes zoster | Pains after meals | Muscular rheumatism |
| | Rubella | Pruritus | Whooping cough | Neuritis | Rubella |
| | Pleurisy | Blurred vision | Neuritis | Pruritus | Cardiovascular disorders |
| | | | Colitis | Vertigo | |
| | | | Haemorrhoids | Epistaxis | |
| | | | Dermatitis | "Writers' cramp" | |
| | | | Dysentery | Blurred vision | |
| | | | Rubella | | |
| | | | Chronic appendicitis | | |

TABLE V

OUTSTANDING NEGATIVE CORRELATIONS BETWEEN PRESENT MENTAL STATE AND DIFFERENTIAL HISTORY PATTERN OF SPECIFIC SOMATIC DISORDERS AS COMPARED WITH CONTROLS

| Psycho-Neurosis | Depression | Schizophrenia |
|---|---|---|
| Peptic ulcer | Nil | Peptic ulcer |
| Constipation | | |
| Diarrhoea | | |
| Haemorrhoids | | |
| Eczema | | |
| Whooping cough | | |
| Dermatitis | | |
| Acne | | |

INFLUENCE OF AGE AND SEX.—Both because of the reasonable assumption that the number of illnesses should increase with advancing years, and because of the low percentage of females in the control group as compared with a fairly even sex distribution in the disturbed psychotic groups, it is of urgent necessity to examine the exact contribution of both these variables to the data.

With regard to *sex*, it should be recalled that no specifically gynaecological complaint or disorder was included in the list investigated, though a differential sex incidence of many specific organic disturbances is, of course, known to occur. In order to smooth out these differences, the total loadings of complaints and diseases of the various psychiatric diagnostic groups (including the controls) were compared for possible sex distinction. A two-way analysis of variance was computed in which

the illness experience of members of the control group and that of patients with neurosis, psychopathy, depression, and schizophrenia were compared, each variable being held separately constant to assess which contributed principally to the variance. Table VI gives the extent of significance of these differences and shows that the combination of diagnosis and sex accounts for 20·5 per cent. of the variance for complaints, and for 52·1 per cent. of actual somatic diseases, whereas psychiatric

TABLE VI

RESULTS OF A PAIRED TWO-WAY ANALYSIS OF VARIANCE TO ASSESS POSSIBLE INFLUENCE OF AGE AND SEX VARIABLES AS COMPARED WITH EFFECT OF PSYCHIATRIC DIAGNOSIS

| Incidence of Somatic Disorders | Combination of Factors Assessed | *F* Ratio | Significance | $\eta^2$ |
|---|---|---|---|---|
| Complaints | Diagnosis plus sex .. .. .. | 33·543 | ·001 | ·521 |
| | Sex only .. .. .. .. | 1·308 | N.S. | ·005 |
| | Diagnosis only .. .. .. | 76·000 | ·001 | ·518 |
| | Addition of sex to diagnosis .. | 2·487 | N.S. | — |
| Diseases | Diagnosis plus sex .. .. .. | 7·959 | ·001 | ·205 |
| | Sex only .. .. .. .. | 3·386 | N.S. | ·012 |
| | Diagnosis only .. .. .. | 16·043 | ·01 | ·185 |
| | Addition of sex to diagnosis .. | 1·379 | N.S. | — |
| Complaints | Diagnosis plus age .. .. .. | 11·174 | ·001 | ·232 |
| | Age only .. .. .. .. | 4·913 | ·01 | ·023 |
| | Diagnosis only .. .. .. | 40·224 | ·001 | ·226 |
| | Addition of age to diagnosis .. | <1 | N.S. | — |
| Diseases | Diagnosis plus age .. .. .. | 41·461 | ·001 | ·529 |
| | Age only .. .. .. .. | 13·127 | ·001 | ·059 |
| | Diagnosis only .. .. .. | 142·590 | ·001 | ·508 |
| | Addition of age to diagnosis .. | 2·249 | ·05 | — |

diagnosis alone accounts for 18·5 per cent. and 51·8 per cent. of the variance respectively. The additional variance obtained by adding the sex variable to that of diagnosis was not statistically significant either for complaints or for diseases.

So far as *age* is concerned, the matter is slightly more complicated, for the neurotics and psychopaths as a group were younger than any of the other diagnostic categories. A similar statistical procedure was however undertaken, a two-way analysis of variance being run on four diagnostic groups (controls, neurotics plus psychopaths, depressives, and schizophrenics) whose separate totals of somatic complaints and

diseases were set out in three age ranges: 15–24, 25–34, and 35–44. As might have been expected, the mean numbers of total complaints and diseases increased progressively as age advanced, but the figures showed that they did so more rapidly in the psychotics than in the controls. As with the variable of sex, while diagnosis and age factors accounted for 23·2 per cent. of the variance with respect to complaints and for 52·9 per cent. with respect to somatic diseases (Table VI), 22·6 per cent. and 50·8 per cent. of this respectively was due to psychiatric diagnosis alone and the addition of age to diagnosis yielded an F ratio which had no statistical significance.

It can therefore be stated that, under the conditions of this investigation, neither the factor of age (within the adult range of 15–44 years), nor that of sex, bore any significance to the somatic illness experience of the subjects assessed relative to the importance of the psychiatric diagnostic classifications in which they were placed.

Estimates of Validity and Reliability.—It is recognized that the findings herein reported, since they are relatively new to psychiatric experience, will require stern tests of their validity. In view of the paucity of similar data elsewhere in the literature, however, it is difficult to assess the results of the present research in terms of strict scientific validity; but it is obvious that if our results are to measure up to certain studies of isolated somatic-disease epidemiology in a psychiatric population, then our control data must reflect reasonably accurately the historical morbidity experience of a representative sample of the British population. It is perhaps unfortunate that such a nation-wide survey does not exist at present. The Registrar-General's annual statistical report relates mainly to mortality figures, and even the incidence of such a widespread notifiable infection as tuberculosis is given either in terms of mortality, or of numbers of notifications by district, or, in other reports, of the relative infectivity of the human or bovine forms or of the radiological incidence of minimal lesions. The writer has not found a recent reference to the historical incidence of the number of individuals who have actually developed tuberculosis *as an illness* over a wide area of the country. Our percentage of 1·9 represents a much lower incidence than that of minimal lesions discovered by mass miniature radiography, and a much higher incidence than the general mortality figures for this disease.

A more comforting indication of the internal reliability of our data is the relative incidence of the acute zymotic infections of childhood. Table I shows that there are no significant differences between the control group and the various psychiatric groups in the historical incidence of measles, mumps, chicken-pox, and scarlet fever. Furthermore the medical history incidence of 74·9 per cent. in the case of measles for the control group is practically identical with that of the Berkeley workers (Bayer and Snyder, 1950), who reported an incidence of 78 per cent. in their longitudinal study of 126 "normal" children followed periodically up to the age of 18 years. Allowing for obvious cultural, climatic, and geographical differences, it is perhaps surprising that the figures for other childhood disorders agree so well. Chicken-pox (71 per cent. as against 42 per cent. in Great Britain) shows considerable discrepancy, but mumps (48 per cent. against 43 per cent.) agrees well, as do rubella (18 per cent.

against 12 per cent.) and diphtheria (4 per cent. against 7 per cent.). Before leaving the Berkeley report, it is interesting to compare figures for other disorders which are close to our own. Thus, for example, the incidence of appendicectomy was 15 per cent. in their study, 14 per cent. in ours; fractures 35 per cent. against 20 per cent.; coughs and colds 67 per cent. against our 52 per cent. Bearing in mind that the two groups studied differed in many respects, were of a different age range, and were many thousands of miles apart, the degree of concordance between the figures is remarkable.

That the figures in the present survey were obtained largely from questionnaire study appears to require some justification, since this method of investigation has been open to considerable criticism in the past. It is of significance however that the morbidity survey conducted by Stocks on behalf of the Ministry of Health (1945) employed a similar methodology of self-assessment and that its validity compared well with non-questionnaire surveys. Analysis of the subjects' statements concerning their health over the previous 3 months revealed that 11 per cent. of the men and 15 per cent. of the women had suffered from nervous or functional disabilities—an incidence practically identical with that reached by Fraser (1947) after very thorough investigation of a group of factory workers. The same report by Fraser's Medical Research Council team contains other interesting comparisons with our findings. For example, his figures (p. 27) show that in both men and women age has relatively little influence on the incidence of either neurotic or physical illness in the range 21–60 years, and also (p. 25) that subjects found to have neurotic illness in industry, not only have a heavier previous history of neurotic breakdowns but also a heavier loading of previous physical illnesses. The M.R.C. Report reveals that 29·6 per cent. of neurotic males and 30·6 per cent. of neurotic females had had previous severe physical illness, while only 20·1 per cent. sympton-free males and 16·8 per cent. symptom-free females had suffered in this way. Both these findings are unexpected, but both are in accord with the results reported here.

As regards the actual differential incidence of various somatic complaints and disorders among the psychiatric groups as opposed to the controls, there are many indications that our findings match the experience of other writers. Thus Wolff (1948) mentions that 8 per cent. of inductees into the U.S. Army complained of recurrent severe headaches, as against 50 per cent. of neurotic soldiers. Our figures (Table I) record percentages of 7·2 and 42·3 respectively for this symptom. Another type of headache, the incidence of which has been accurately determined in an American population, is migraine. Lennox (1941) gives the incidence of migraine in 1,000 otherwise healthy medical students and nurses as 5·4 per cent. Our figure of 8·2 per cent. in the control group compares reasonably well, especially as the comparable incidence among cases of psychoneurosis, anxiety state, is so widely different at 37·7 per cent.

A report of the Ministry of Health (1922) on the incidence of rheumatic diseases mentions that 2·76 per cent. of the total list strength attended their practitioners for "rheumatic disease" in 1922. This is a vague diagnosis, but our control figures for certain forms of non-articular rheumatism agree well with this estimate (*e.g.* lumbago

2 per cent.; fibrositis 3 per cent.). The results when psychiatric patients' histories are considered are very different. Harris (1938) in a 10 to 12-year follow-up study of 259 cases of neurosis, anxiety state, found that 10 per cent. of 98 cases available for study had suffered from muscular rheumatism, an estimate which may be compared with our figure of 12 per cent. for controls and 18 per cent. for anxiety states. Harris also found 6 per cent. of his cases with essential hypertension (present series: 5 per cent. anxiety states, 10 per cent. controls); 4 per cent. with arthritis (present series: 3 per cent. anxiety states, 5 per cent. controls); and 21 per cent. with gastric symptoms ("gastric stomach" in present series: anxiety states 22 per cent., controls 3 per cent.).

Finally, it is relevant to refer to another study (Eysenck, 1947) in which the incidence of somatic complaints in cases of anxiety state is compared with that in cases of hysteria. Analysis of responses to a psychological test for "neuroticism" (Maudsley Medical Questionnaire) showed that anxiety-state cases answered "yes" more often than hysterics in most cases. The same tendency was present in our series. Out of 45 comparable disorders, anxiety states showed a higher percentage incidence in 32, as against hysterics who showed a higher percentage incidence in thirteen.

## Discussion

The high incidence of somatic complaints in psychiatric patients has been referred to by several authors, including Billings (1939) and Hemphill (1939), but the efforts of Ross and others (1950) to assess the concomitance of mental disorder and such somatic vegetative diseases as peptic ulcer, asthma, diabetes mellitus, rheumatoid arthritis, and hypertension, lead to the conclusion that none of these disorders presented more commonly among the population of a Canadian mental hospital than in the general population at risk outside it. For purposes of comparison with our present findings, however, it is significant that the Canadian workers were evaluating the *concurrence* of mental and physical disease, and that, in so far as their findings included observations that peptic ulcer and rheumatoid arthritis at least were *less* common among psychotics than among the general population, their results are in accord with our thesis. The conclusions regarding rheumatoid arthritis confirm those of Gregg (1939). It appears that the response to stress involves the total personality: the sphere of major response may be somatic, with resulting physical disease; it may be psychological, and followed by psychiatric disturbance; or it may partake of both with the development of both somatic and psychic disorders, either together or in alternation.

The literature on immunity has concerned itself little with these considerations, yet certain indications that a case can be made out for the implication of psychological factors in disease susceptibility and host resistance are beginning to appear. A recent paper by Vaughan (1949) testifies to a decreased ability on the part of 22 schizophrenic subjects to develop antibody titres to inoculated pertussis vaccine antigen as compared with seven control subjects. Not only were the maximal titres of the schizophrenic group significantly lower than those of the controls, but they also showed a differential distribution among the various sub-types of schizophrenia studied, paranoid and better-integrated schizophrenics giving relatively higher titres than constitutionally

determined and less well-integrated hebephrenics. That these findings are not peculiar to the inoculation of pertussis vaccine or to a schizophrenic population has already been shown (Lovett Doust and Chappell, unpublished observations). Similar experiments were performed using the H and Vi antigens of the Lister Institute T.A.B. vaccine on more acute and less chronic institutionalized schizophrenics and also on patients with other psychiatric disorders. In schizophrenia, lower absolute titres were found than in controls; in affective disorder, depressive type, the titres were higher but still not up to control levels; and in cases of psychoneurosis, initial post-inoculation titres were equivalent to those in controls, but were ill-sustained and fell off markedly during a 10-week observation period.

If such antibody responses can be held to represent certain aspects of the immunity threshold, then it may be said that psychiatric variables are at least of equal importance with those dependent upon adequate nutrition, since Gell (1948), studying titres obtained after the inoculation of starved Wuppertal Concentration Camp prisoners in Germany with a complex group of provocative antigens, found differences between the results from this group and from healthy, well-nourished British soldiers which only just reached the level of statistical significance. In view of these figures, Gell was forced to the conclusion that even gross, pre-mortal undernutrition "does not play as large a part in widespread epidemics as is generally supposed", and hence that the "psychological imponderables" may be assumed to be a responsible influence. These psychological imponderables might also go a long way to explain the wide variations in susceptibility which exist among healthy individuals, a fact which still puzzles immunologists (Beattie and Dickson, 1943).

ALTERNATION OF PSYCHO-SOMATIC DISEASES.—It appears to the writer an attractive hypothesis that a man's individual constitution is a limiting factor with respect to the degree of stress which he can withstand. If it may be assumed that breakdown follows exposure to stresses which go beyond the individual's threshold of tolerance, then not only is it logical to include in such stresses their emotional and psycho-dynamic components, but it is also just as likely that the resulting total personality breakdown may predominantly affect the psyche as it is that the soma may be principally involved. Disease and disorder may conveniently and with some truth be thought of as homeostatic defences against the onslaught of stress. The schizophrenic's withdrawal from intolerable stress into a dream world of relative unawareness is, at least temporarily, as successful for him as it is socially morbid for his place in society; the "mentally stable" patient who is incapacitated by acute stress and spends a month away from his work with pneumonia, and the recurrent invalidism necessitated by the chronically summating stresses leading to recurrent exacerbations of rheumatoid arthritis, are equally morbid in so far as they solve a temporary situation on a basis of somatic pathology which often becomes permanent and irreversible.

Mental and physical diseases alike are both pathways of reaction and defences against stress. The greater the mental stability and balance, the more likely is the psychiatric pattern of breakdown to be chosen in preference to the "canalization" and

"somatization" of breakdown into tissue and organ systems. The psychiatric investigation of cases of somatic disease (*e.g.* Alexander, 1950) shows that under such somatic defences there lie conflicts and profiles which are also common in many respects to specific organic disease conditions, and Appel and Rosen (1950) have illustrated the alternation from psychiatric to somatic disturbance and *vice versa* which is obvious in so far as it is looked for. Indeed it may be said with Hippocrates (Aphorisms, VII, 5 and VI, 21) that the one orientation of disease often acts as a protection against the other. Clow and Prout (1946) have recently reported amelioration of mental disorder in cases in which intercurrent physical disorder appeared. Of their 100 unselected mental hospital patients who experienced a physical illness during their period of hospitalization, they found that 67 improved and eleven recovered completely.

Our previous findings suggest that, since the medical histories of psychiatric patients show a greater loading with somatic disease than do those of a comparable healthy control group, then either their experience of stress has been greater or their resistance to it is intrinsically lower than that of the controls. Probably both factors operate and, since initial breakdown predisposes to other breakdowns, we may assume a vicious-circle mechanism.

PHYSICAL DISEASE THE MASK OF PSYCHOSIS.—A further implication of these results lies in the fact that, in historical sequence, it is usually the physical illness mechanism of defence which is first employed by the subject under stress. That psychiatric breakdown is a last defence, resorted to when other means have failed, would seem to be a function of the degree of emotional stability and maturity achieved by the subject (Draper and others, 1946). Psychiatric investigation of apparently mentally stable patients with such psychosomatic diseases as asthma, essential hypertension, and peptic ulcer, reveals a chaos of repressed and unconscious conflicts, tensions, and desires for which the organic disease is often an obvious symbolic means of expression and of partial detumescence. Too rapid and dramatic a removal of somatic symptoms (*e.g.* by ACTH in rheumatoid arthritis) not uncommonly leads to a concomitant psychiatric disturbance or fully-fledged psychosis. It is suggested therefore that the physical illness, determined as it is by the combination of type of stress and personality pattern, is both the mask and the means of expression for the tensions and conflicts which, if uncanalized and unexpressed, would amount to a psychosis.

## SUMMARY

(1) The physical illness history, in terms of 38 bodily complaints and 72 somatic diseases, was assessed in 354 healthy adult controls and in 272 patients with a variety of psychiatric disorders.

(2) It was found that the physical illness experience of the psychiatric patients was significantly in excess of that of the controls, psychotics having a heavier loading of previous physical illness than neurotics or psychopaths. The influence of age and sex was insignificant in comparison with that of psychiatric diagnosis.

(3) The childhood illness experience in all groups did not differ significantly with respect to most zymotic diseases. A differential pattern of organ system reaction was

found to be present for other disorders. The somatic illness history of neurotics included many examples of minor physical disability, that of depressives showing especially a loading with gastro-intestinal disorders, and that of schizophrenics a loading with locomotor and cardio-vascular diseases.

(4) The findings are discussed with reference to their reliability and validity, and their immunological implications considered from the psychiatric point of view.

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# SPREAD OF INFLUENZA IN A FACTORY

BY

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It is generally agreed that colds, influenza, and other respiratory ailments are among the principal causes of sickness absence in industry, and that some of this absence results from cross-infection between fellow-workers. The total working days lost far exceeds those lost for other reasons, such as industrial disputes, yet comparatively little study has been devoted to the subject of infection in factories. Experiments with ventilation equipment, ultra-violet irradiation, etc., are considered too costly, too time-consuming, or too difficult to assess. Thus the factors which favour or restrict infection at work are not precisely known. In theory, it should be possible to learn a great deal from statistics of sickness absence. In practice, sickness absence rates are so profoundly influenced by the age and sex of the workers and the season of the year that valid comparisons between departments or factories require more elaborate standardization than existing records allow. An even more serious defect is that the records rarely include sufficient data about such conditions of work as are likely to affect morbidity.

The difficulty of standardization can be by-passed in a study restricted to rates of absence from influenza, since this disease does not discriminate between men and women, affects all age-groups, and tends to occur in relatively short and well-defined epidemics. If the records also include a nucleus of environmental data, some of the laws governing cross-infection at work may be elucidated.

This paper describes the use made of an opportunity provided by the 1951 influenza epidemic. The material collected was too slight to support any original methods of analysis, but it did provide an interesting sequel to a previous study of the spread of tuberculosis in factories (Hewitt and Stewart, 1951).

## Material

On January 12 it was reported that a considerable number of cases of influenza were occurring amongst the employees of the University Press, Oxford. One of us (F.A.) then began a series of visits to the factory in the course of which she interviewed every employee who was absent from work for any cause at any time between December 1 and February 15. For all persons who were absent the following information was obtained: first day of absence; day of return to work; reason for absence.*

Those who were diagnosed as cases of influenza were also asked whether they had had home contact with the disease before falling ill, whether they had obtained a doctor's certificate, and when exactly they had sickened. Much additional information was also collected by observation and from factory records, including the number of persons working in each room, the floor measurements of each room with a plan of the layout, and the ages of all employees.

* The mere statement by a worker that he did or did not have influenza was not accepted without satisfactory confirmation from answers to other questions. The points regarded as essential to the diagnosis were coryza, pyrexia, "aches and pains", depression, and lassitude.

When the offices as well as the workshops were included, there was found to be a total of 51 occupied rooms, containing 752 workers. During the span of the epidemic (December 27 to February 2), there were 145 cases of influenza with no recurrences, an overall incidence of 19·3 per cent. During the same period, 141 persons recorded one or more absence from causes other than influenza. The distribution of these absences by cause and by sex is shown in Table I.*

TABLE I

DISTRIBUTION OF ALL ABSENCES BY CAUSE AND BY SEX OF WORKERS

| Sex | Male | Female |
|---|---|---|
| Incidence of influenza (all cases) | 17·8% | 23·1% |
| Incidence of cases with mention of home contact | 3·9% | 3·8% |
| Average number of working days lost per case of influenza | 5·4 days | 5·4 days |
| Incidence of absence from all other causes | 15·1% | 27·2% |
| Average number of working days lost per absence from all other causes | 5·2 days | 3·7 days |

Judging by the average number of working days lost, the disease appears to have taken a milder form than in the contemporary epidemic at Widnes and Runcorn reported by Potter (1951), where the average duration of absence was stated to be 14 to 21 days.

The average number of days lost in Oxford was exactly the same for men as for women, and the rather greater case incidence amongst women is not statistically significant.

In only one-fifth of the cases did questioning elicit any mention of a home contact with influenza before the development of symptoms.

A more detailed analysis of the absences due to influenza is given in Table II.

TABLE II

DISTRIBUTION OF INFLUENZA ABSENCES BY AGE AND SEX

| Sex | Age Group | Cases of Influenza | Estimated Population at Risk | Estimated Incidence of Influenza (per cent.) |
|---|---|---|---|---|
| Male | Under 25 | 27 | 120 | 22·3 |
| | 25–44 | 26 | 193 | 13·5 |
| | 45 and over | 43 | 227 | 18·9 |
| Female | Under 25 | 35 | 154 | 22·7 |
| | 25–44 | 12 } 14 | 46 } 58 | 24·1 |
| | 45 and over | 2 } | 12 } | |
| All Groups | | 145 | 752 | 19·3 |

A $\chi^2$ test was used to compare the numbers of cases observed and the numbers of workers estimated to have escaped influenza with the numbers expected on the hypothesis that risk of infection is independent of sex and age. This gave a value of $\chi^2$ equal to 7·05 which, with four degrees of freedom, lies between the 20 per cent. and 10 per cent. points of $P$, *i.e.* the observed variations between sex and age groups are not statistically significant.

The result of applying a statistical test to Table II is to show that it does not contain evidence of any significant variation in incidence between the sexes or between the age groups

* The sex of workers was only recorded during the survey for those persons who were absent. The incidence rates of influenza and other absences in Tables I and II are therefore based on the numbers of each sex working in the factory in December, 1950. This qualification does not apply to the other incidence rates quoted in this paper, nor does it affect the average number of days lost per absence.

considered. This is in accord with the observation of Padley (1947) . . .

> Influenza shows a fairly level relative incidence throughout life . . . There is in these figures no indication of a developing immunity against influenza . . .

Consequently no standardization of incidence rates has been considered necessary in the following arguments despite the fact that the groups of workers considered do not have identical sex and age composition. It is probable that the appearance of variation in Table II is a result rather than a cause of the statistically significant variation in Table IV.

## Spatial Distribution of Cases

In addition to the written records, a plan was kept of each room in which influenza occurred, on which the working place of each victim was marked and dated. There was an appearance of "clumping" in some of the rooms which suggested an association between exposure to draughts and liability to develop influenza. But when the timing of the cases was considered it was found that the plans did not provide any evidence for a chain of infection linking successive cases or generations of cases. This was not taken to mean that no cross-infection had been taking place, for two reasons:

(i) Different powers of resistance in individual workers, together with the operation of chance factors, would make the visible distribution of cases very different from any invisible chain of infection which might exist.

(ii) It had been shown in the previous study of tuberculous infection that no contradiction arose from assuming that *all* workers in the same room are within infecting range of each other, and not merely those who work close together. That this may hold true for influenza as well as tuberculosis is suggested by Table III.

If proximity within a room is an important factor in determining the risk of cross-infection, then rooms where workers are closer together should, other things being equal, show a higher incidence of illness than rooms in which they are further apart. In Table III each room for which floor measurements were obtained has been allotted to one of three groups defined by the ratio of floor area to number of occupants.

Some of the minor rooms and offices with twelve occupants or less were not measured and are therefore omitted from Table III. Altogether in these omitted rooms there were 118 workers with thirteen cases of influenza. Maintenance workers (37 workers with ten cases of influenza) are also excluded from Table III since their duties involved periods of work in several different rooms.

TABLE III

Incidence of Influenza related to Spacing of Workers

| Range of Area-per-worker (sq. ft) | No. of Workers in such Rooms | Cases of Influenza | Incidence per cent. | Average Area-per-worker (sq. ft) |
|---|---|---|---|---|
| 0 to 99 | 207 | 50 | 24·2 | 76 |
| 100 to 199 | 216 | 39 | 18·1 | 128 |
| 200 or more | 174 | 34 | 19·5 | 301 |
| All Measured Rooms | 597 | 123 | 20·6 | 160 |

To test the hypothesis that the incidence of influenza was not affected by the amount of area-per-worker, a $3 \times 2$ contingency table was formed of the numbers of workers in each group who did and who did not fall ill. This gave an insignificant value of $\chi^2$ (2·58 for two degrees of freedom, $P > \cdot 20$).

The differences in incidence which appear to be associated with differences in area per worker are well within the range of chance variation, and there is no evidence that the risk of infection was associated with the distance between workers. It might be argued that area per worker is an inadequate index of the proximity of workers. But it was found that attempts to measure mean linear distance between workers, or to substitute airspace for floor-space, begged many questions and settled none. Since neither the room plans, nor the analysis in Table III revealed any tendency for infection to pass more easily between the closest neighbours than between more widely separated workers, it has been assumed that at any given moment risk of infection is the same in all parts of a room.

### Relation of Infection Risk to Number of Contacts

Each room in the factory was placed in one of three groups according to the number of workers it contained, and the influenza experience of the three groups is arranged for comparison in Table IV. The maintenance workers are again excluded because they cannot be allotted to any particular room or group of rooms.

TABLE IV

Incidence of Influenza related to Number of Workers per Room

| Workers per Room | Number of Workers in such Rooms | Cases of Influenza | Incidence per cent. |
|---|---|---|---|
| 1–15 | 197 | 23 | 11·7 |
| 16–75 | 308 | 65 | 21·1 |
| 76 or more | 210 | 47 | 22·4 |
| Total | 715 | 135 | 18·9 |

When the number of workers in each of the three room-groups who did or did not fall ill, was arranged in a $3 \times 2$ contingency table, a value of $\chi^2 = 7 \cdot 58$ was obtained for two degrees of freedom. The corresponding value of $P$ is between 2 and 3 per cent. which denotes significant association between number of workers per room and the incidence of influenza.

The result of applying a statistical test to Table IV is to show that the risk of influenza was definitely lower in the small rooms of the factory than in the larger rooms. Sex and age differences—themselves not of significant magnitude—cannot be responsible for this result, since the small rooms in the factory contain a relatively large proportion of women who, in the factory as a whole, had a slightly higher incidence than men. The proportion of men belonging to the age group with the lowest incidence was no greater in these rooms than in the factory as a whole. It may be alleged that a higher employment status or "sense of responsibility" could reduce the number of cases of sickness absence in office workers. To this there are two answers. In the first place, when the factory is divided by size of room, as in Table IV, many of the junior clerical staff are found in the smallest rooms, while the largest rooms contain most of the highly-skilled and best-paid workers. In the second place, the illnesses diagnosed as influenza in this study reduced sufferers to a condition in which the effort of brainwork became intolerable. A piece of evidence which reinforces our interpretation of Table IV is provided by the proportion of cases in which a home

contact with influenza was reported: 8 out of 23 in the small rooms, 21 out of 112 in the remainder of the factory. This means that 35 per cent. of the cases in the small rooms compared with only 18 per cent. in the large rooms had a known previous contact with the disease at home.

In short, low incidence seems to have been characteristic of the small rooms as such, not the result of any incidental circumstance. This justifies the belief that in each room the risk of influenza was related to the number of potential sources of infection in the room. It is interesting to note that the observed difference in incidence occurs chiefly in passing from the first to the second group of rooms in Table IV, while the increase between the second and third is negligible. This "early saturation" feature accords with what was to be expected (on the basis of the theory advanced in the earlier paper) of a disease spread by a relatively large number of infectious persons. This point is developed in the discussion below.

## Distribution of Cases in Time

Table V shows the distribution of cases according to the day of the week on which sickening occurred. The shape of the epidemic curve was such that, if there had been no weekly pattern of infection, each day total (composed of six individual days cases) would have the same expected value of 20·9.

TABLE V

Frequency of Influenza Cases According to Day of Sickening

| Sunday | Monday | Tuesday | Wednesday | Thursday | Friday | Saturday | Total |
|---|---|---|---|---|---|---|---|
| 19 | 22 | 20 | 19 | 19 | 33 | 13 | 145 |

If, however, the aggregation of workers in a factory constitutes a special infection risk, then on Monday, when workers return to the factory from the relative seclusion of the week-end, more persons should be infected than on any other day. Provided also that there is some well-defined incubation period, one day of the week should have a particularly heavy case incidence. In fact, the figure for Friday in Table V is suggestively high—and the figure for Saturday suspiciously low.

A $\chi^2$ test of the departure from equal daily frequencies gives a value of $\chi^2 = 10 \cdot 7$ with 6 degrees of freedom, which falls just beyond the 10 per cent. level of significance.

Although no weekly cycle of infection can be proved from Table V, the apparently well-defined maximum occurring on Fridays would be consistent with such a cycle, and with a modal incubation period of four days. It is of interest to compare this result with the report of Hope Simpson (Bradley, 1951) who found no modal interval between first and second cases of influenza in the same family.

## Discussion

It seems clear that influenza, like tuberculosis, is a greater hazard to the factory worker when he is employed in a large room than when he is employed in a small one. But it does not follow that the relation between risk of infection and numbers of

workers can be formulated in the same way for these two very different diseases. In fact, the task of assessing the risk of influenza presents a more difficult problem.

First we may restate the theory of the previous paper on tuberculosis. We employ the following notation:

$(U+1)$=the number of people in a special group which is sufficiently compact for a specified infection to pass from any one member to any other. Examples might be the group of persons using a certain swimming bath, or the workers in a particular workshop. Each member has $U$ contacts within the group who may or may not be infectious. He also has, as a member of the general community, a further number of contacts which we call $D$. It is assumed that, in any sufficiently large group, the average value of $D$ will be the same as in the community of which that group forms a part.

$M$ = the percentage of a group of persons who will develop a disease within a specified period under conditions of maximum exposure to (*i.e.* certainty of) infection. This depends on the resistance of the group to disease in general as well as the degree of acquired immunity.

$\pi$ = the proportion of the members of a community who are carriers of a disease, or who are infectious at some time during the period.

$y$ = the expected percentage incidence of a disease during the period for which $M$ and $\pi$ have been defined.

Then we have

$$y = M\left\{1 - (1 - \pi)^{D+U}\right\}$$

The expected incidence, $y$, is an increasing function of $(D+U)$ with a maximum value, $M$, which it approaches at a speed determined by the "carrier rate", $\pi$.

If infectious persons are as rare as in the case of tuberculosis, there will still be important increases in $y$ as $(D+U)$ passes from 200 to 300 and beyond. But when they are as common as the sources of influenza virus during an epidemic, then $y$ will, for all practical purposes, be at its maximum before $(D+U)$ reaches 50. This offers an explanation of the relatively minute difference between the incidence in the second and third groups of Table IV, and suggests that the highest observed incidence lies near $M$. We may therefore take 23 per cent.* as a reasonable estimate of $M$. That is to say, just under a quarter of this factory population were susceptible to attack during the epidemic.

Some idea of the average carrier rate can also be obtained. For present purposes a "carrier" may be defined as a person who, although beginning to sicken for influenza, is nevertheless present in the factory. The number of workers whose answers to questions indicated that they had been carriers in this sense was 44, which, divided by the total population at risk, gives an estimated value to $\pi$ of 0·057 for the factory as a whole.

* When the maintenance workers, who move from room to room through the factory, are added to the third group in Table IV we have: workers, 247; cases, 57; incidence, 23·1 per cent.

In the tuberculosis study, values of both $\pi$ and $D$ were obtained from data concerning morbidity in rooms of different sizes by means of the estimating equation given above. This involved making two assumptions which seemed fair in the case of the tuberculosis study but are not of general validity. In the first place it had to be assumed that sources of tuberculosis infection were evenly distributed throughout all sizes of workshop in the industrial community studied. At the very beginning of an influenza epidemic, infectious persons may well be distributed at random, in which case the equation will describe the relation between risk and size of room. But if cross-infection is taking place this random distribution will only apply during the first generation of cases. During the second and successive generations of the epidemic the proportion of infectious persons will be higher in the large than in the small groups. That is, instead of being a constant, $\pi$ will itself become an increasing function of $U$, and the estimating equation cannot be used to obtain estimates of $\pi$ unless $D$ is already known.

The second assumption of the tuberculosis study concerned the duration of exposure to infection. Here the main source of infection which had to be considered was the worker in whom the disease might pass unrecognized, or be ignored for months and sometimes for years. During this time he would distribute sufficient bacilli to cause disease in any other regular occupant of his workshop who was susceptible. But the sources of influenza virus were workers sickening for an illness which would cause them to withdraw from the factory very quickly, possibly before the room as a whole had been infected. In rooms where this happened the expected incidence would be lower than $M$ by an unknown amount. Only in rooms which contained a succession of infectious cases could we be at all confident that every potential victim had fallen ill.

This discussion illustrates the need for more study of the way in which patterns of infection at work may be represented. An improved model of the factory epidemic should dispense with the two assumptions here discussed. It should also treat the epidemic as a "stochastic" process (Bailey, 1950), avoiding the determinism of the usual over-simplified treatment. On the other hand, it should be simple enough to use for estimating purposes or it will give no practical guidance on the likely results of this or that policy of control.

The only obvious way in which a factory epidemic of the kind described could be controlled is by reducing the carrier rate. If it were possible to keep sickening workers out of the factory, or to persuade them to leave as soon as they began to feel ill, some advantage could be gained. This is especially difficult in the case of a disease which reaches a highly infectious stage without warning, besides which any system of sponsored absence from work would be open to abuse. But two assertions may be made with reasonable confidence:

(i) It is not necessary to accept the pessimistic view of Potter (1951) that

> It is probable that any method intended to obviate or reduce the risk of becoming infected at work will have little effect in the total incidence of the disease.

For if the risk of infection in the larger rooms of this factory had only been

reduced to the level of the risk in the small rooms, then about half the cases of influenza would have been prevented, and this would have been a *net* reduction in total incidence.

(ii) Little short of the complete exclusion of infection could have prevented the generation of *some* new cases in rooms with very many workers. A comparatively modest programme of control would, however, be a paying proposition in factories where the rooms are all fairly small.

An attempt must be made in some future study to relate varying sickness incidences within and between factories to other environmental factors which may facilitate or hamper the passage of upper respiratory infections between workers. Among these atmospheric temperature in the workshop has a claim to early investigation.

## Summary

(1) An epidemic of influenza in a factory is described and analysed. It is found to reproduce two features of infection in workshops previously observed in the case of tuberculosis:

(*a*) Risk of infection is not related to the proximity of workers within the rooms.

(*b*) Risk of infection is related to the number of workers in a room.

The form of this relation suggests that, for the purpose of respiratory infection, each worker may be regarded as in effective contact with every other regular occupant of his room.

(2) Some of the difficulties of setting up a theoretical model for a factory epidemic are discussed.

Our thanks are due to the Printer to the University and to the employees of the University Press, in particular the staff of the Personnel Department, whose generous co-operation made this study possible. We also wish to acknowledge the help we have obtained from our colleagues Dr. Josefine Webb and Dr. Alice Stewart.

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# BOOK REVIEWS

**Demographic Yearbook 1949-50, Second Issue.** 1950. United Nations, Department of Social Affairs (Population Division), Department of Economic Affairs (Statistical Office). New York. Pp. 558. (45*s.*; $6; *Sw. Fr.* 24).

With the publication of the first issue of the Demographic Yearbook, a precedent was established in the presentation of comprehensive and sound demographic data for almost every country in the world. The most reliable and recent figures on population, mortality, natality, and migration were collected for the years up to and including 1947, making possible up-to-date descriptions of each country and comparisons of characteristics in different countries.

This second issue of the Yearbook continues the main descriptions and further enlarges the field covered in the first issue. It gives a number of basic tables of population composition, economic characteristics, mortality rates, and migration across national boundaries for the years up to 1948 and in many cases up to 1949. Various sources of data are used, but for a number of the tables special questionnaires were answered by government and administrative departments. Undoubtedly the population data thus presented constitute the most comprehensive and up-to-date body of knowledge at present available. Moreover, this issue includes in the text a chapter which analyses regional population trends and attempts some interpretation of them.

In addition to the basic tables of the first issue, several new tables are included, some of which will be repeated annually, and others every five years. The subject of special emphasis in this issue, that of marriage and fertility, is one of particular interest to the western countries with their ageing populations. The special tables here presented cover a wider field than the customary summaries of marriage and fertility rates: there are tables of fertility rates by age of father, of births by age of mother and order of birth, and of information concerning surviving children, proportions of children under 5 years of age, and the like.

In such a large array of tables from countries differing so widely in their methods of collecting and recording data, it is hardly surprising that discrepancies become apparent—due in some cases to the divergence of ideas and definitions in different countries, and in others to inaccuracies of all kinds. These irregularities are discussed in the text at the appropriate points, and certain steps have been taken to minimize them; moreover, several devices are introduced to safeguard against misinterpretation of the data. One of these devices in connection with population data, takes the form of a code describing the type of estimate from which the data were obtained and indicating the reliability of the estimate.

Nevertheless, the non-comparability of figures for many countries detracts to some extent from the value of certain comparisons. In every table curious differences between countries emerge, and it is important to know whether these represent real differences or whether the curious points are only the results of inaccuracies in figures or different definitions of categories. For instance, Table 6, which gives the percentages of unmarried men and women in each age-sex group, reveals a very wide range of differences. France apparently has very small proportions of single men and women at the higher ages, whilst Ireland stands out as having large proportions of unmarried men and women at all ages and particularly at the higher ages and above 65 years. Other points of interest are disclosed in other tables: from Tables 5, 7, and 8, which refer to marriage status and to women by age and number of

children, a great divergence of marriage and family patterns is revealed. In Canada the numbers of children per 1,000 women are greater than in most countries, and are especially high for young mothers; in Switzerland, the numbers of children are low, but not for older mothers. Again, in the U.S.A. and in the Netherlands the numbers of children per 1,000 women of all ages are very similar, but in the U.S.A. a clear pattern of young marriages and early family-building is indicated, whilst in the Netherlands the numbers of children born to older mothers are consistently high. The whole set of tables on marriages, births, and fertility is extremely valuable, and the linking of these tables leads to many interesting conclusions—if the figures can be relied upon.

The set of tables on economic characteristics discloses other curious facts, such as the very high proportion in Japan of women over 65 years of age who are economically active. This apparently high proportion may depend on the definition of "economically active", which would be different in industrial and in agricultural countries and could lead to false conclusions, especially in respect of the female population.

In each succeeding issue, the Demographic Yearbook will continue to bring the basic tables up to date and will select a new subject for special emphasis, the information being directly collected for this purpose. The reports, tables, and summaries thus presented will be of permanent value to local and national authorities concerned with health, employment, population, and other social responsibilities. The successive issues will build up an encyclopaedia of knowledge to which every social scientist, educationist, and student of world affairs, should have access.

LILLI STEIN

**Probation and Related Measures.** United Nations Department of Social Affairs. New York, 1951. Pp. 284, with seven appendices and bibliography. H.M.S.O., London. (22*s.* 6*d.*)

This is an important study, well documented, and collating data from many countries. It is the first book on the prevention of crime and the treatment of offenders to be published by the United Nations Secretariat.

The aim of probation is the social rehabilitation of offenders without punishment. It usually means the conditional suspension of punishment after trial and sentence, and the personal supervision and guidance of the probationer. It is recognized in most civilized countries that short-term sentences do nothing to "cure" the early criminal but merely bring the law into disrespect, and that treatment should fit the offender rather than the offence. Probation is thus in no sense a " let off ", but punishment is suspended and may be imposed at any time during the period of surveillance if the conduct of the probationer so deserves. "Commutation" and "pardon" must both be distinguished from probation, for in these conditions the prisoner is not automatically subjected to personal supervision. Parole, the supervision of pre-delinquents and other maladjusted persons, binding-over, and conditional suspension of sentence without probationary supervision, are all designed to reduce or avoid prison sentences, but differ from probation in the absence of personal contact between the probationer and his supervisor.

In most countries probation was introduced by statute, but in England and in the State of Massachusetts, U.S.A., it developed from common law practice.

It is a common belief that probation is extended only to juvenile offenders facing their first conviction. This may have been true in the past, but in most countries to-day, age and previous convictions are no bar to probation. Serious crimes, and those carrying heavy *minimum* prison sentences, are by law exempted; otherwise the personal qualities of the prisoner and the chances that he will benefit from probationary supervision are the deciding factors.

Probation may be initiated by two different legal procedures. The Anglo-American method provides for the suspension of the punishment after sentence, whereas the procedure obtaining in most countries of Europe suspends the sentence itself, since it is felt by many that the

salutary effect of a formal conviction and verbally-expressed sentence may be discounted by the crippling effect in after-life of a recorded conviction.

The best way to provide the close, personal relationship which is the basis of probation is to select and train suitable probation officers with the greatest care. The work being still ill-paid and the profession largely unrecognized, there is a lack of volunteers. The administration of the probation system differs among nations. In Great Britain the responsibility rests with the local authority, and there is no official recognition of the work of voluntary individuals and religious bodies. In the Netherlands, on the other hand, while probationary supervision is a function of the department of the public prosecutor, the actual work of probation and social rehabilitation is carried out by volunteers and religious bodies, both subject to a considerable measure of state control.

It is generally considered that probationary supervision can best be provided by a large voluntary body with a small nucleus of trained personnel. Whichever method is used, there is little doubt that probation, by permitting the offender to remain a useful member of the community, offers an alternative to imprisonment which is highly desirable in a modern society.

CATHERINE SWANSTON

**UNESCO: Report of the Co-ordinating Committee on Abstracting and Indexing in the Medical and Biological Sciences.** 1951. Pp. 92. Imprimérie Bellenaud, Paris; H.M.S.O., London. (5*s.*)

This report covers the origin and work of the committee from the preparatory conference at Unesco House in October, 1947, to the end of 1949.

**Supplement [1951] to the Catalogue of Economic and Social Projects, 1950.** United Nations Publication Sales No. 1951. II.D.2. Pp. 310+xiv. H.M.S.O., London. (17*s.* 6*d.*)

This supplement to the "Catalogue of Economic and Social Projects 1950" covers the work in progress during 1950 and carried over or approved for initiation in 1951. It reflects the 1951 programme approved on January 1, 1951, and should be used in conjunction with the 1950 Catalogue which remains the basic document.

# ABSTRACTS

*This section of the* JOURNAL *is devoted to selected abstracts of articles on social medicine appearing in the current literature. The section will be edited in collaboration with the three abstracting Journals,* Abstracts of World Medicine, Abstracts of World Surgery, Obstetrics, and Gynaecology, *and* Ophthalmic Literature.

**Tuberculosis in Medical and Nursing Students.** BATES, R. C., and DAVEY, W. N. (1951). *Amer. Rev. Tuberc.*, **63,** 332. 3 figs, 9 refs.

Student nurses and medical students at the University of Michigan were skin-tested with purified protein derivative (P.P.D.) and *x*-rayed at 6-monthly intervals. Two doses of P.P.D., of 0·0001 and 0·005 mg., were given intracutaneously. In students in whom the tuberculin reaction was "converted" to positive, chest radiographs were obtained at 3-monthly intervals for 2 years thereafter. Active pulmonary tuberculosis was considered to be present in any tuberculin-positive student who had *x*-ray evidence of a lesion in the lung which was interpreted by the clinician and radiologists as compatible with tuberculous infection.

Results were reported covering a 10-year period for student nurses and a 3-year period for medical students. No evidence of a decreasing incidence of tuberculin sensitivity in succeeding classes was found, nor could any one school year be shown to be associated with increased hazards of infection. Twenty cases of active pulmonary tuberculosis were detected and all but two were minimal when first discovered. In no instance had the lesion progressed under observation to more serious disease, and the infection had relapsed in only one patient to date. All cases of active tuberculosis appeared in students who had been negative to 0·005 mg. of P.P.D. on entrance to the school. *M. Daniels*

**Paralytic Poliomyelitis following Injury.** HARRINGTON, A. B. (1951). *Lancet*, **1,** 987. 6 refs.

Some 100 unselected cases of poliomyelitis occurring in the Forces were observed, and in five of these a definite association with trauma was noted. The paralysis appeared in from 2 to 10 days following the trauma, and in each case there was a relationship between the muscles affected and the site of injury. *T. Anderson*

**Report of an Investigation into an Outbreak of Poliomyelitis in a Small Town Remote from Melbourne.** MCCLOSKEY, B. P., (1951). *Med. J. Aust.*, **1,** 612.

The author describes two cases, both in infants, out of eight notified cases of poliomyelitis. The two infants, 9 and 11 months of age, had been immunized within the preceding 2 weeks with a combined diphtheria toxoid and pertussis vaccine. The paralysis affected the limb into which the injection had been given. He suggests two possibilities: either that the babies were already suffering from a subclinical infection or that they had been contaminated by means of a syringe after use on another affected subject. *Franz Heimann*

**Tonsillectomy and Poliomyelitis. I. Studies on Incidence in 1949.** SIEGEL, M., GREENBERG, M., and MAGEE, M. C. (1951). *J. Pediat.*, **38,** 537. 6 figs, 20 refs.

Advantage was taken of the 1949 epidemic of poliomyelitis in New York to investigate the relationship between this disease and tonsillectomy: 2,446 cases with 179 deaths occurred in a population of approximately 8,000,000. By applying the age-specific morbidity experience of the general population it was possible to compare the morbidity experience of the tonsillectomized with that of the non-tonsillectomized group. In spite of the small number of tonsillectomized patients who subsequently contracted the disease, the conclusion appears justifiable that tonsillectomy predisposes to the development of clinical symptoms of poliomyelitis. For cases occurring within one month of operation this risk appeared several times greater for the tonsillectomized than for others of comparable age. *W. G. Harding*

**II. Frequency of Bulbar Paralysis, 1944–1949.** SIEGEL, M., GREENBERG, M., and MAGEE, M. C. (1951). *J. Pediat.*, **38,** 548. 1 fig., 24 refs.

In this paper the clinical types of poliomyelitis encountered in post-tonsillectomy cases are investigated. The expected incidence of bulbar paralysis is estimated from the experience of over 6,500 cases of poliomyelitis which occurred in New York City between 1944 and 1949, and a comparison is also made between tonsillectomized cases and others individually "paired" with them in respect of age, sex, date of onset, and residential area. Both methods show a significant preponderance of bulbar paralysis in the post-tonsillectomy group, with a maximum incidence within the first 3 weeks after operation. This persisted for 6 months after operation, but was not statistically significant after the first month. The case fatality was also higher in the post-tonsillectomy group. *W. G. Harding*

**The House Fly as the Chief Factor Responsible for Infant Mortality from Gastro-Intestinal Disease.** (La mosca domestica principale responsabile della mortalita' infantile per malattie gastroenteriche). CORBO, S. (1951). *Riv. Parassit.*, **12,** 37. 4 figs, 6 refs.

The author compares the infant mortality rates from gastro-intestinal diseases in two areas of Latina: in Zone A, where insecticides have been used against the mosquito; and in Zone B, which has not been sprayed until recently. The following table shows the figures obtained:

| Year | Zone | Insecticide | Infant Mortality (per 1,000 live births) |
|---|---|---|---|
| 1945 | A | None | 31·33 |
| | B | None | 17·79 |
| 1946 | A | DDT. | 7·74 |
| | B | None | 11·71 |
| 1947 | A | DDT | 18·00 |
| | B | None | 12·18 |
| 1948 | A | DDT+Octachlor | 11·59 |
| | B | None | 13·27 |
| 1949 | A | DDT+Octachlor | 3·36 |
| | B | None | 11·40 |
| 1950 | A | DDT+Octachlor | 8·10 |
| | B | DDT+Octachlor | 5·16 |

The striking fall in mortality in Zone A in 1946 and 1949 and in Zone B in 1950 is believed to be due largely to the disappearance of flies; the rise in Zone A in 1947 in spite of DDT is attributed to the occurrence of DDT resistance. An increase in morbidity rates from gastro-intestinal disease in certain selected areas also coincided with increases in the number of flies in those areas.

*A. Paton*

**Homologous Serum Hepatitis and Measles Prophylaxis. A Report to the Medical Research Council.** COCKBURN, W. C., HARRINGTON, J. A., ZEITLIN, R. A., MORRIS, D., and CAMPS, F. E. (1951). *Brit. med. J.*, **2**, 6. 23 refs.

Homologous serum jaundice is rare in children, and case fatality rate varies with different batches of icterogenic material. During an investigation in 1949 into the value of gamma-globulin fraction of human plasma in the prevention and attenuation of measles in susceptible contacts, a group of ten children were given varying doses of a batch (LP 309) of dried plasma, reconstituted in a small volume of fluid, instead of the gamma globulin itself. Subsequently seven developed homologous serum disease, of whom three died. Encephalopathic symptoms were present in some cases, and in one without clinical jaundice.

The plasma pool LP 309 came from sixty donors, of whom six had a history of jaundice 5 to 40 years previously; the remaining 54 had no such past or subsequent history. Such information as was derived about their other donations of blood did not show any of the six to be carriers of the virus. Part of this plasma pool had been processed for gamma globulin and nominated LG 309. It was possible to trace 43 known and 29 possible recipients of LG 309. One of the known recipients developed mild jaundice, but none of the others was affected.

*J. F. Loutit*

**The Prevention of Whooping-cough by Vaccination.** WHOOPING-COUGH IMMUNIZATION COMMITTEE OF THE M.R.C. *Brit. med. J.*, **1**, 1463 (1951). 1 fig., 31 refs.

This paper describes the trial of pertussis immunization carried out by a committee of the Medical Research Council. Children between 6 and 18 months whose parents agreed to innoculation were allocated by random selection to a "vaccinated" and an "unvaccinated" group. The former received injection of pertussis vaccine; the latter received injections of an "anticatarrhal" vaccine specially prepared and bottled so as to resemble the pertussis vaccine. Five different pertussis vaccines were used; three were plain suspensions and two were alum-precipitated. Ten trials were conducted in five separate districts in England.

The vaccinated group contained 4,515 children and the unvaccinated, 4,412; of these, 3,801 and 3,757 respectively completed the prescribed course. Contact with some of the children was lost (mainly through removal) so that at the end of the investigation the totals (in the same order) were 3,358 and 3,352. The trials were begun in 1946 and completed in 1950. The period of observation in each group, was on the average, 27 months. A composite chart is given which shows that the period of observation in each area of study covered periods of increased incidence of pertussis.

Details are given of a large number of attributes of the two groups, and these all serve to show that there was a close similarity between them. So far as reactions to the vaccination were concerned, there was little to comment upon in regard to the plain vaccines; six children given alum-precipitated material developed sterile abscesses. In particular, it is perhaps valuable to state that there were no convulsions or evidence of encephalopathy and that no child developed poliomyelitis within 2 months of inoculation. [In the latter case, the risk of a child developing the disease was probably remote.]

The attack rates per 1,000 child-months were 1·45 in the vaccinated and 6·72 in the unvaccinated, a ratio of 1 to 4·6. In both groups the attack rate in females was higher than that in males. Two forms of exposure were analysed—"home" and "other". In the former group the attack rates were 18·2 per cent. (vaccinated) and 87·3 per cent. (unvaccinated). In the latter group the respective figures were 8·3 per cent. and 38·0 per cent. It is of interest that the ratios of the incidences in home, other, and "unknown" exposures were very similar—namely, 1 to 4·8, 1 to 4·6, and 1 to 5·0.

When the severity of the attack in each group was analysed, it was found that 73 per cent. in the vaccinated group were mild; the corresponding figure in the unvaccinated was 24 per cent. So far as duration of immunity could be estimated, it seemed that there was no falling off in the protection afforded. Post-nasal swabs were taken in 96 per cent. of the clinically diagnosed cases. From 60 per cent. of them *Haemophilus pertussis* was isolated, *Haemophilus parapertussis* was isolated from 24 children, and *Haemophilus bronchisepticus* from one child.

[This is a first-class piece of work which must be read by all interested in the subject, as an abstract can do no more than select some of the more important comparisons. It provides an example of how such a study should be planned and carried out.] *T. Anderson*

**Diurnal Variations in Body Temperatures.** MELLETTE, H. C., HUTT, B. K., ASKOVITZ, S. I. and HORVATH, S. M. (1951). *J. appl. Physiol.*, **3**, 665. 2 figs, 25 refs.

Continuous rectal temperature determinations were made on eleven male and eleven female subjects, all healthy adults, by means of copper-constantan thermocouples and an automatic recording potentiometer. Four 24-hour studies showed that almost all maximum and minimum temperatures occur between 6 p.m. and 6 a.m., and therefore principal emphasis was placed on the night hours. There were a total of 42 such 12-hour observations. Maxima for males averaged 38·32° C. (100·98° F.), at 8·49 p.m. and for females 38·36° C. (101·05° F.), at 10·49 p.m.; minima, males 36·83° C. (98·29° F.), at 6·04 a.m. and females 37·16° C. (98·89° F.), at 4·48 a.m. With the exception of the levels of the maxima, these values were significantly different between the two sexes. One male showed only 0·70° C. (1·26° F.) total range throughout the night, while one female showed as much as 2·08° C. (3·74° F.). Fifteen minute-averages were plotted against time and each graph fitted with the best possible trend line by a modified method of least squares. The mean rate of fall from evening to morning was −0·12° C. per hour (0·22° F.) for males and −0·06° C. per hour (0·11° F.) for females, a significant difference.

Deviations from each trend line, namely shape of the temperature curve, were examined for possible relationship to time, evening meal, cold refreshments, activity, falling asleep, awakening, or changes in ambient temperature. Although the inflexions were distributed apparently at random, it was found that of the twelve graphs demonstrating upward inflexions, seven began within 15 minutes of the onset of moderate activity. In general sleep had a stabilizing effect on the shape of the temperature curve.—[Author's summary.]

**The Effect of Tobacco on Lung-volume.** WHITFIELD, A. G. W., ARNOTT, W. M., and WATERHOUSE, J. A. H. (1951). *Quart. J. Med.*, **20**, 141. 13 refs.

This paper comes from the Department of Medicine and Medical Statistics of the University of Birmingham. The authors determine the total lung volume and all its subdivisions in 58 healthy male subjects, and correlated the results with their present and past consumption of tobacco. There was a uniform tendency for the correlation coefficients of the vital capacity, complemental air, and reserve air to diminish, and for the functional residual air and residual air to increase, as smoking increased. The residual-air increases were statistically significant. There were also increases in the ratio of residual air to total lung volume. These changes were reversible, and ventilatory efficiency improved when the habit was given up. Effects were much more in evidence when recent tobacco consumption had been high. Smoking also produced a diminution in chest expansion. *I. Ansell*

**Cardiovascular Disease in Cotton Workers. I.** SCHILLING, R., and GOODMAN, N. (1951). *Brit. J. industr. Med.*, **8**, 77. 7 figs, 22 refs.

The authors discuss the high death rates from cardiovascular disease among cotton workers. Analysis of the Registrar-General's occupational mortality data shows an association, in the three occupational groups in the industry, between mortality and exposure to dust. Strippers and grinders, the group with the highest exposure to dust, have a significantly high mortality from cerebral vascular lesions, which supports the theory that hyperpiesis may be caused by it. Analysis of data obtained from death certificates is rendered difficult by the fact that up to 1939 the Registrar-General gave priority to cardiovascular disease over respiratory disease when death was certified to be due to multiple causes, so that in heart disease coexisting with the occupational disease byssinosis the former cause was automatically selected. Moreover, practitioners may make use of the convenient diagnosis of myocarditis more frequently than is strictly warranted. Even after allowing for these potential sources of error, however, there seems to remain a significant excess of deaths due to cardiovascular disease in strippers and grinders.

*W. G. Harding*

**Left-Handedness in Children.** (Recherches sur la gaucherie infantile). SCHACHTER, M., and COTTE, S. (1951). *Praxis*, **40**, 195. 14 refs.

Among 4,200 patients visiting a neuropsychiatric guidance clinic there were 150 left-handed children, and 100 of these were examined more closely. The time of dentition, of walking, and of speech development was normal. The development of the intellect (Binet–Simon) did not deviate from the average for the entire group. Only 70 per cent. of these children had a history of normal birth; enuresis, nail-biting, and speech and character disturbances were more frequent among them than among the entire group. In 23 per cent. of the cases there was left-handedness in the family; 30 per cent. of the parents were alcohol addicts and 46 per cent. of them were mentally disturbed. *O. Budde-Goppingen* [*Excerpta Medica*]

**Hearing and Speech Problems in Children. Observations and the Use of Electroencephalography.** MARCUS, R. E. (1951). *Arch. Otolaryng., Chicago*, **53**, 134. 1 fig., 14 refs.

It is most important that hardness of hearing should be detected as early as possible, for the child's entire social development depends on beginning the education as soon as possible, preferably at about 3 years of age; also it is most important that the parents should be prepared as soon as possible for the need of special education. In a series of 71 patients, four showed Rh incompatibility; in another one this was doubtful. The diagnosis was made on: (*a*) a history of neonatal jaundice; (*b*) blood sampling showing the mother Rh-negative and father and child Rh-positive. Cases where the father was Rh-negative and mother and child Rh-positive were not included. In six cases maternal rubella in the first 3 months of pregnancy was the only positive factor in the history. One mother had rubella in the seventh month, but this is of doubtful importance. There were two cases of birth injury, six of premature

birth, five cases among twins, six cases of infantile infectious disease, and 45 of "other or unknown causes". There was no case of consanguinity and no case of proved syphilis.

The great difficulty in testing children for deafness is that under 5 years of age children give unreliable responses to pure-tone tests, which do not hold interest or attention, and the various conditioning tests are not usually performed reliably under two years of age. The use of the electroencephalograph in the sleeping child is therefore suggested, either during the afternoon sleep or under the influence of a mild barbiturate. When the child is asleep the mother calls its name, with increasing loudness. In nineteen out of 53 children with severe hearing loss there was electroencephalographic evidence of intra-cranial disorder, and also in nine of eighteen children with speech defects and slight to moderate hearing loss. *F. W. Watkyn-Thomas*

**Familial Idiopathic Methaemoglobinaemia.** BREAKEY, V. K. ST. G., GIBSON, Q. H., and HARRISON, D. C. (1951). *Lancet*, **1**, 935. 2 figs, 9 refs.

Two cases of methaemoglobinaemia in a brother and sister are described. In both the cyanosis responded to the oral administration of methylene blue followed by ascorbic acid. A number of the relatives of these patients were examined and were all found to be free from methaemoglobinaemia, and the view is expressed that familial methaemoglobinaemia is inherited as a recessive character. The assays of diaphorase I (co-enzyme factor I) showed a deficiency in activity in both the patients as compared with normal controls. In the first case the capacity of the intact cells to reduce methaemoglobin in the presence of glucose and lactate in the absence of methylene blue is impaired as compared with a control. *Geoffrey McComas*

**Congenital Exstrophy of the Urinary Bladder: Late Results of Treatment by the Coffey-Mayo Method of Uretero-Intestinal Anastomosis.** HARVARD, B. M., and THOMPSON, G. J. (1951). *J. Urol.*, **65**, 223. 2 figs, 20 refs.

In 1925 Charles Mayo stated that the survival rate of all persons with congenital exstrophy of the bladder was 50 per cent. at their tenth year and 33·4 per cent. at their twentieth year. [Coffey's original paper on uretero-intestinal and choledocho-intestinal anastomosis was published in 1911.] This survey shows how the position has improved. Of 144 cases treated by uretero-intestinal anastomosis at the Mayo Clinic between 1912 and 1946, the survival rate was 74 per cent. at the tenth year and 52 per cent. at the twentieth year. At 25 years the survival rate was 50 per cent. (of a total of 28 cases), and at 30 years four out of eight were alive.

The operative mortality rate for a two-stage cystectomy was 10·5 per cent. Eleven cases had had a one-stage operation, with a mortality of 36 per cent. The cause of death in hospital was either pyelonephritis and obstruction at the anastomosis (55 per cent.) or a leaking anastomosis (22 per cent.); the commonest cause of death after leaving hospital was pyelonephritis with stones or hydronephrosis, or both, in 65·7 per cent.

In 41 cases excretory urograms were recorded at various periods after operation; in only five were they considered normal. In four cases renal calculi were present. Dilatation of the kidneys and ureters was equally common on the two sides, but non-visualization was found to be more common on the left side.
*K. Whittle Martin*

**Sickle-cell Anaemia and Sickle-cell Trait as Homo- and Heterozygous Gene-combinations.** LEHMANN, H. (1951). *Nature, Lond.*, **167**, 931. 18 refs.

In America and in Italy evidence is accumulating to show that subjects with the sickle-cell trait are heterozygous, and those with sickle-cell anaemia homozygous, for the gene which determines sickling. In a population in which a gene has a frequency $p$, the fraction of heterozygotes is $2p(1-p)$ and of homozygotes $p^2$, so that if the anaemia is not lethal, $p^2+2p(1-p)$ should give the fraction of the population which carried one or more of the genes under consideration; thus, if 43 per cent. of a population are found to have one or more factors for sickling, about 6 per cent., or one-sixteenth, should be homozygous and 37 per cent. heterozygous.

Certain East African tribes have an incidence of sickling of this order of magnitude. The Lango, a Nilotic tribe, have an incidence of about 37 per cent. sickling in subjects of all ages from below 3 months to old age. The pygmoid Baamba have a 45 per cent. incidence of sickling. Such high percentages show that homozygotes must number between 5 per cent. and 10 per cent., unless the homozygous state causes death *in utero* or shortly after birth. The author comments: "Both are events which could not have escaped the attention of medical officers in African maternity wards", especially since in Hamitic tribes the incidence of sickling is low—below 1 per cent. In one kraal in which there was close intermarriage, there were 46 individuals with sickle cells out of a population studied of 53.

The author concludes that the simple hypothesis that heterozygotes manifest the sickle-cell trait and that homozygotes manifest the anaemia is not in accordance with the facts. He favours Raper's suggestion that the development of the anaemia may depend on some factor imported by mating with persons other than Africans—a hypothesis which would explain the appearance of the disease in Indians, Arabs, Italians, and American negroes, and in its absence from pure Africans.

[An alternative suggestion would be that the Bantu have accumulated a number of "modifier" genes which suppress the effect of the sickling gene when in the homozygous state, and that mating with other races breaks up the assembly which protects homozygotes from ill effects. Such "epistasis" is well recognized. In dogs, for example, the cross Newfoundland × Great Dane gives rise to grossly abnormal offspring because in the Newfoundland a group of modifier genes protects against the ill effects of genes which produce pituitary hyperfunction (see Huxley, *Evolution*). Nevertheless Foy and Kondi (*E. Afr. med. J.*, 1951, **28**, 1) report four cases of sickle-cell anaemia among thirteen mothers and infants of the Jaluo tribe whose cells showed sickling.]
*G. Discombe*

**The Heredity of Epilepsy as told by Relatives and Twins.** LENNOX, W. G. (1951). *Amer. med. Ass.*, **146**, 529. 5 figs, 16 refs.

The incidence of epilepsy among the parents, sibs,

and children of 4,231 epileptic patients was studied. Among the relatives, anyone subject to seizures not considered to be due to syncope, hysteria, carotid sinus syndrome, or the result of "defective metabolism" was classified as epileptic, irrespective of whether any precipitating causes, such as alcoholism or fever, were also present. The question whether one or two seizures were necessary for the diagnosis was avoided by accepting one seizure as sufficient in one-half the relatives and two seizures in the remaining half. The initial series of patients was divided into two groups according to whether or not there was evidence of cerebral damage antedating the first seizure; 22·9 per cent. gave a history of antecedent gross brain lesions, and of their close relatives 1·8 per cent. were found also to be epileptic. Among the close relatives of the remaining 77·1 per cent. of patients who were classified as having "essential" epilepsy there were 3·6 per cent. similarly affected. No very satisfactory information was available to show the incidence of epilepsy (using the name criteria of diagnosis) in the general population. However, it was found that in the two world wars approximately 0·5 per cent. of all recruits were excluded because of epilepsy. This undoubtedly represented a minimum figure for the incidence of the condition in the general population, because the screening was itself fairly coarse, and also because it did not include persons confined to institutions. Thus at the most the incidence was only 7·2 times greater among the relatives of the epileptics than in the general population.

A series of 122 twin pairs (69 monovular and 53 binovular), one at least of each pair being epileptic, was also studied. Of the monovular twins 61 per cent. were both affected, whereas among the binovular twins only 9 per cent. were both affected. Furthermore, it was found that in those pairs of identical twins where both were epileptic there was a closer resemblance between the members of each pair, in respect to the clinical character of the epilepsy and the electroencephalographic findings, than would have been expected on a chance basis.

While it is concluded that hereditary factors are important in determining predisposition to epilepsy, it is emphasized that genetic prognosis can be advanced only very cautiously and must always involve a detailed consideration of the situation in the particular family concerned. *Harry Harris*

**Stenosis and Imperforation of the Rectum.** (Sténoses et imperforations du rectum). NUBOER, J. F., and AKEN, H. VAN (1951). *J. Chir., Paris.*, **67**, 209. 10 figs, 1 ref.

The embryological development of the imperforate anus is discussed and four types are recognized: anal stenosis, anal atresia, anal and rectal atresia, and rectal atresia. The third type, where there is no anal canal and the termination of the rectum is a considerable distance from the skin, is unfortunately far the commonest. This type is generally associated with a persistent fistula between the rectum and the genito-urinary tract. Thirty-five cases are described, of which 24 were of this type; in 22 of these there was an associated fistula. Of these fistula thirteen were recto-vaginal and eight, occurring in males, were recto-vesical. The picture which appears in most text-books showing a blind pouch with a fistula leading off one side is quite incorrect. The fistula is, in actual fact, the funnel-shaped end of the rectum, and the pouch is due to ballooning of the side of the rectum from pressure of faeces. The authors are opposed to early radical surgery for two reasons: (1) 30 per cent. of patients have additional congenital defects, usually cardiac —until it is known whether they are likely to survive there is no point in performing extensive surgery; and (2) the mortality of early radical surgery is unjustifiably high. If the condition of the baby is good a colostomy is performed; if the condition is bad a caecostomy is carried out. Nothing more is done until about the age of 6 years. The rectum is then completely mobilized through a sacral approach, and the funnel-shaped fistula brought out through the anal dimple. Should faecal incontinence follow, a fascial sling repair (Wreden-Stone) is performed. There have been nine deaths among 35 patients treated; only one patient has been left with incontinence, the results otherwise being satisfactory. *R. T. Burkitt*

**Pseudocyesis : a Psychosomatic Study in Gynecology.** FRIED, P. H., RAKOFF, A. E., SCHOPBACH, R. R., and KAPLAN, A. J. (1951). *J. Amer. med. Ass.*, **145**, 1329. 21 refs.

Physical and psychiatric examinations were carried out on 27 patients (23 negro and four white) with pseudocyesis. The age range of the patients was 18 to 36 years; 22 of the 27 were admitted to the Endocrine Clinic of the Jefferson Hosptial, Philadelphia, between July, 1946, and January, 1949; the proportion of cases of pseudocyesis among admissions to the maternity clinic was 1 in 250. The patients were observed over 1 to 3½ years, and 36 episodes of pseudocyesis were recorded; the number of episodes in individual patients ranged from 1 to 4; eleven had never actually been pregnant.

The first and commonest symptom present in 26 patients, was a menstrual disturbance: hypomenorrhoea in nineteen and amenorrhoea in seven. A history of gradual abdominal enlargement was given by 24. Inversion of the umbilicus was not seen. Breast changes were frequent: enlargement, tenderness, secretion of milky or cloudy fluid, and enlargement of Montgomery's tubercles and the areola. Foetal movements were reported by 22 patients, as early as the first month and as late as the eighth month. These usually took the form of quivering or pulsating sensations in the left upper quadrant of the abdomen. Softening of the cervix was observed in nineteen patients. Gain in weight was generally greater than in normal pregnancy. Nausea, frequency of micturition, salivation, sleepiness, and faintness were often reported. "Labour" occurred in two patients at the expected date, but ceased abruptly when the women were told that they were not pregnant. The authors point out how easily a mistaken diagnosis of pregnancy may be made on superficial examination: nine of the 27 had been told by one or more physicians that they were pregnant.

Although sixteen patients had been pregnant before the pseudocyesis, only one had a living child, and the desire for a child was strong in every patient in the series. Contributory motives were: (1) the need to secure the husband's wavering affections; (2) the need to prove the ability to conceive and to achieve parity with other women; (3) the wish for a child as a plaything or companion; and (4) the need for self-punishment. As a group, the patients were gullible and insecure; personal

relationships were unsatisfactory and frustration-tolerance was low.

Laboratory studies, including endometrial biopsy, estimation of urinary gonadotrophin and 17-ketosteroids, and examination of vaginal smears, were carried out on some of the patients. The results of these studies may be summarized thus: ovarian function was good, and there was evidence of persistence of corpus luteum activity. This could account for many of the clinical manifestations, such as menstrual disturbances and softening of the cervix. It is presumed that mental conflict is capable of influencing the pituitary and causing the release of lactogenic hormone, the suppression of follicle-stimulating hormone, and consequently persistence of luteal function. The abdominal enlargement is due to fat and forced contraction of the anterior abdominal muscles.

The key to treatment is psychotherapy. Telling the patient bluntly that she is not pregnant is to be condemned: she may threaten suicide, or go from one physician to another seeking a diagnosis of pregnancy. Gland treatment of itself is unlikely to prevent recurrences unless the patient is given some insight into the nature of her condition. Treatment is directed towards helping the patient to recognize and face her conflict and to deal with it in a more effective way. *Desmond O'Neill*

**Psychological Investigation of Senile Dementia.** DÖRKEN, H., and KRAL, V. A. (1951). *Geriatrics*, **6**, 151. 1 fig., 26 refs.

Believing that some of the organic psychoses show specific differences in response to the Rorschach ink-blot test, the authors analysed the Rorschach responses of 35 hospital patients in whom the diagnosis was senile dementia. Only those patients over 65 years of age were selected who had no significant localized brain lesion and had no history of mental illness or deficiency, were sufficiently co-operative, and could answer correctly at least one item on the Wechsler-Bellevue adult intelligence test. The patients were presented with all ten Rorschach cards and attempted all the items of the full intelligence test, whence an indication of their emotional and intellectual deterioration was obtained. The findings of the investigation were compared with similar work by Prados and Fried, Klopfer, and Piotrowski on cases of senile dementia, normal senescent decline, and organic brain damage respectively.

On the basis of this comparison and the analysis of their own results the authors draw these main conclusions: (1) The process of deterioration bears no necessary close relationship to age. (2) Reduction of capacities—such as an unduly long reaction time—and deterioration in personality structure, as exemplified by a high percentage of pure form and absence of colour, shading, and movement responses indicative of general inadequacy and loss of individuality rather than of "rigid control" of thought are related to the extent of the dementia. (3) An "efficiency quotient" of 50 to 55 is apparently a crucial level, at which complete inability to respond appropriately (gross inaccuracies of form perception) and a serious loss of contact with reality first appear. (4) The process of deterioration, as seen in the Rorschach and Wechsler results, is selective in its destruction of emotional and intellectual factors. (5) Certain striking pathological elements in the responses to the Rorschach cards, such as frequent "original" and "bizarre", as opposed to "popular", responses, perseveration, confabulation, oligophrenic detail, and certain accepted "signs" of organic brain damage, indicate that the test may be useful in differentiating senile dementia from normal senescent decline, late paranoid reaction, and depression. *N. A. Standen*

**Induced Abortion.** (Der provozierte Abort). KAYSER, H. W. (1951). *Arch. Gynäk.*, **179**, 451. 12 figs, 25 refs.

Of 625 cases of abortion admitted to hospital in Kiel in one year, 153 were induced. In 88 per cent. the abortion followed an intrauterine injection, usually of soap solution, and in 12 per cent. there had been some form of intra-uterine manipulation. Over half the patients were married and no difference was noted between these and the single patients in the timing of the abortion.

The preference for soap solution appears to be due to its rapid action, half these cases aborting within 20 hours; water took 50 hours, and intra-uterine manipulation 100 hours. The danger of soap is its infective nature and its local corrosive action; necrosis of the cervix was seen in some cases and in two cases the unusual complication of infection of the right sacroiliac joint was seen.

Treatment of the infected cases was along the usual conservative lines, penicillin and sulphonamides being used, and only one patient died. *D. C. A. Bevis*

**Life Situations, Emotions, and Nasal Disease. Evidence on Summative Effects exhibited in Patients with "Hay Fever".** HOLMES, T. H., TREUTING, T., and WOLFF, H. G. (1951). *Psychosom. Med.*, **13**, 71. 2 figs, bibli.

The burden of this paper is that the reaction of the nose to mixed ragweed pollen, in both sensitive and non-sensitive persons, is influenced by the state of the nasal tissues at the time of exposure. When the nasal mucosa is already in a condition of hyperfunction, such as occurs in infective rhinitis and in various emotional states, the response to pollen, as shown by hyperaemia, hypersecretion, swelling of the mucosa, and pain and itching in the nose, is greater. Experiments are described to illustrate these reactions.

Stimulation of anxiety and resentment in a patient who had hay-fever, without exposure to pollen, was shown to produce nasal obstruction and discharge, sneezing, and itching in the nose with pain. Another patient was exposed to pollen during a psychiatric interview. The effect on nasal function was slight until the interview was directed towards the patient's personal conflicts; symptoms and signs then increased considerably and did not diminish before the patient had been reassured and had recovered her mental balance.

The character of the mucous-membrane response in the hay-fever syndrome appears to be determined by: (1) the intensity and duration of exposure of sensitive individuals to the specific allergen; and (2) the intensity and duration of other environment threats and assaults which provoke a response of nasal hyperfunction. In the latter group are situations arousing an emotional reaction which is reflected in the state of the nasal mucosa.

*Desmond O'Neill*

**Life Situations, Emotions, and Exercise Tolerance.** DUNCAN, C. H., STEVENSON, I. P., and WOLFF, H. G. (1951). *Psychosom. Med.*, **13**, 36. 10 figs, 10 refs.

The authors set out to determine the relation between emotional tension, cardiac efficiency, and the occurrence of symptoms on exercise. Their subjects were eleven patients with such symptoms as palpitation, dypsnoea, and dizziness, but no organic heart disease; sixteen patients with these symptoms and organic heart disease; and eight healthy controls. Each subject was given a full physical and psychiatric examination. Measurements of circulatory function were made before and after exercise. Cardiac output and stroke volume were estimated by the ballisto-cardiograph; the cardiac index was defined as the output in litres per minute per square metre of body surface.

In the controls there were fewer emotional changes and less variation in exercise tolerance (E.T.) than in the patients. In all subjects, however, on the days of impaired E.T. there was awareness of mild psychic tension which prevented relaxation. On such days the cardiac indices at rest were higher than on the days of comparative relaxation; the higher output was due mainly to an increase in stroke volume rather than in heart rate.

In the subjects who had a long-standing intolerance of effort it was found that the signs and symptoms accompanying effort varied with the degrees of stress imposed by the circumstances of the moment. It was possible to show that a reduction in stress and therefore in emotional tension was followed by an improvement in circulatory function, as indicated by stroke volume and cardiac index. A similar close relationship between changes in E.T. and in the emotional state was observed in the patients with organic heart disease. In these the impairment of E.T. was made up of an organic element and a functional element. In a heart much damaged by disease mild emotional tension could cause symptoms of effort; anxious patients were intolerant of effort even if organic damage was not severe. In some of the patients studied over a period of time, symptoms of effort intolerance were shown to be due to cardiac over-activity related to environmental stress and not to structural heart disease, and when emotional tension was relieved symptoms disappeared and cardiac efficiency increased.

E.T. was tested in several subjects before and after rapid changes in emotional tension which were experimentally induced. A reduction in tension following ventilation of feeling was found to be associated with an objective improvement in E.T.; where the interview increased anxiety and resentment, E. T. was impaired. In general there was a correlation between complaints of palpitation, dyspnoea, and dizziness and the degree of impairment of E.T. Symptoms occurred in some patients with intense bodily preoccupation during only mild physiological disturbances.

An important part of the treatment of a patient with heart disease is limitation of physical exertion. Equal importance should be given to reducing the load placed on the heart by cardiovascular mobilization associated with anxiety and resentment due to life stress; the increased cardiac work which this causes may be not only as great as that imposed by everyday effort, but also much more prolonged, and indeed in some patients almost constant. *Desmond O'Neill*

**Psychiatric Indications for Termination of Pregnancy.** (Die psychiatrischen Indikationen zur Unterbrechung der Schwangerschaft). BINDER, H. (1951). *Schweiz. Arch. Neurol. Psychiat.*, **67**, 245. 20 refs.

In contrast to the dogmatic condemnation of abortion in all cases, the medical view is that abortion is justified if the life of the mother is in danger or her health cannot be maintained by any other means. The decision whether such prerequisites exist is difficult and controversial even when physical lesions are present: it is more difficult in presence of physical deviations and here a generally acknowledged code of guidance is missing.

The author cites certain mental defects (*seelische Fehlentwicklungen*) as indications for the termination of pregnancy, including the following: chronic depressive states increasing irritability and outbursts of anger, asthenic, hysterical, hypochrondriacal, or paranoic developments, the anxiety-complex, and organ-neurosis. As conditions whereby, in the presence of such defective development, the health of the woman may be crippled permanently and profoundly are mentioned: a chronic conflicting situation, abnormal mental sensibility, lack of resistance, especially when these conditions existed before pregnancy, and when, if pregnancy continues to term, serious conflicting situations are to be expected. The mental state at the date of examination is not so decisive as the whole development of the woman's life. Sometimes, in spite of tempestuous mental reactions, the interruption must be refused because the woman has a normal psychic constitution.

Suicidal attempts are rare; only 3 per cent. of all suicides are pregnant women. Thus the danger of suicide is insufficient to justify interruption. To prevent suicidal attempts the author recommends temporary treatment in an institution; this operates favourably in women who were mentally healthy before the pregnancy. but adversely when the woman was previously mentally unbalanced. The consent of the parents or, in the case of a married woman, of the husband should always be obtained. There is sometimes danger that the termination of pregnancy may later on cause a form of "abortion neurosis".

Psychosis *per se* is not an indication for inducing abortion. It is justified only if there is danger that the pregnancy and its consequences may result in grave and permanent worsening. In cases of schizophrenia there is as a rule no change for the worse; it is an indication only if the symptoms occur for the first time during pregnancy or if symptoms previously present grow rapidly worse. Epilepsy is an indication if the frequency of fits increases at the beginning of the pregnancy.

The course of all other psychoses, especially in the manic-depressive patient, is independent of generative processes and in most cases there is no indication for abortion.

When, in an insane woman, the termination of pregnancy is recommended, the sterilization of the patient should be performed at the same time since each following pregnancy entails the danger of deterioration. In women who are not self-reliant sterilization may have to be ruled out either on account of the later mental upset or

because the woman may perhaps later bear a child without great risk to her mental health if she is guided by an intelligent, appreciative husband.

The psychiatric indications for termination of pregnancy must always depend to a certain extent on subjective judgements. *O. Burger*

**A Statistical and Clinical Study of Sarcoma of the Uterus.** (Statistische und kasuistische Beiträge zum Sarkom des Uterus). PLENGE, K., and FALGE, E. (1951). *Z. Geburtsh. Gynäk.*, **134,** 145. 11 figs, 19 refs.

Statistics of uterine sarcoma do not possess the uniformity associated with figures published on carcinoma. Improved methods of diagnosis and the wide appreciation of the importance of the subject have provided abundant material for investigation of both corporeal and cervical carcinoma, and figures have become more or less standardized; but with uterine sarcoma statistics show a remarkable variation. The proportion of sarcomata to myomata, for example, varies in literature from 0·3 per cent. (Raab) to 10 per cent. (Werner); the present authors seek an explanation for this divergency. They consider that the number of cases recognized is small because of difficulty in diagnosis, and quote Mayer, who said that, if tissue thought to be myomatous were more carefully examined, in an increasing number of instances it would be found to be sarcomatous. An enormous amount of time and trouble is required for serial section and staining, but success would often follow.

A further difficulty is that no common criteria have been laid down as to what histologically constitutes a sarcoma. Mistakes and uncertainty are admitted by many authors. Hüssy had a 39-year-old unmarried patient who, 2 to 3 months after extirpation of the uterus for myoma, developed a recurrence which proved to be a myo-fusicellular sarcoma. Nasnata found a sarcoma in a cervical stump 5 years after a hysterectomy for myoma microscopically confirmed to be benign. Boschann discovered retroperitoneal metastases in a patient whose uterus had been removed 9 years before for myoma; these were described as borderline.

Material from two hospitals in Berlin was sent to the present authors. This series extends over 5 years (1945–9); 713 primary tumours were studied from one hospital, of which 371 were benign myomata, 310 carcinomata, and 32 sarcomata (23 intramural and nine endometrial). From the other hospital during the same years 497 tumours were examined; there were 264 myomata, 213 carcinomata, and twenty sarcomata (fourteen intramural, three endometrial; two were double tumours, in one case an intramural sarcoma with an endometrial sarcoma, and in the other case an intramural sarcoma with a carcinoma corporis). Altogether 3·4 per cent. of all uterine tumours examined by the authors were sarcomata.

[The original paper should be studied by the interested reader.] *E. W. Kirk*

**The Psychoprophylactic Method of Painless Labour.** (Психопрофилактическое обезболивание родов) SHYSHKOVA, V. N., BRONSHTEIN, R. M., and IVANOVA, E. I. (1951). *Akush. Ginek.*, **No. 2,** 25.

Under the system described by the authors the expectant mother is enlightened about the physiology of pregnancy and labour in a series of sytematic lectures; towards the end of pregnancy the conduct of labour is repeatedly demonstrated to the patient.

This method was applied to 107 expectant mothers and painless labour was observed in 80·4 per cent. of the cases. The analysis of less successful cases showed that the number of instructions (lectures) was directly proportional to the efficiency of the method. It was also important that the last instruction should be given not more than 5 to 6 days before the date of expectant delivery. *E. W. Collis*

**Experimental Application of the Psychoprophylactic Method for the Conduct of Painless Labour.** (Опыт обезболивания родов психопрофилактическим методом) KORABELNIK, B. K., DARON, D. I., SIERDUKOVA, O. G., MELEROVICZ, E. E., and MUSATOVA, N. I. (1951). *Akush. Ginek.*, **No. 2,** 29.

This method was applied to 316 expectant mothers, of whom 204 were primigravidae and 112 multiparae. The sources of the fear experienced by women in labour were determined and analysed. The origin of fear may be unknown or may derive from impending labour pains, the possibility of perineal tears, and fear of foetal or maternal death. All types of fear could be removed by appropriate instructions.

In this series of cases 85·3 per cent. of primigravidae and 88·4 per cent. of multigravidae experienced excellent or good results in achieving painless labour. The weight of the infant had no influence on the conduct of labour or upon the degree of pain suspension. Many patients started labour at home; hence the assessment of the duration of labour could not be accurate. The foetal mortality was 0·95 per cent.; this mortality rate could be further reduced.

The psychoprophylactic method is regarded as harmless, securing painless labour at all stages of parturition, and seems to have no contraindications. *E. W. Collis*

**Conduct of Painless Labour by Means of Suggestion.** (Обезболивание родов внушением без предварительной гипнотической подготовки) KOGANOV, M. I. (1951). *Akush. Ginek.*, **No. 2,** 31.

This method consists of conversation with the patient which starts at the time of admission and proceeds until confinement to bed in the labour ward is completed. Throughout this time the patient is instructed that the labour shall be painless and each step to accomplish it is explained.

The labour ward is situated away from the main hospital and lit by pleasant, warm light; silence is particularly observed. With each oncoming uterine contraction the patient is instructed to shut the eyes and proceed to sleep.

The effects observed on 460 patients in labour are said to be very encouraging; in 87 per cent. of the cases there was completely painless labour. There was no difference between the groups of primiparae and multiparae. The younger group of patients, between 26 and 30 years, showed somewhat better results than the group

with an average age above 30. This method was applied to expectant mothers who did not receive antenatal psychoprophylaxis and reported to hospital directly in labour.

*E. W. Collis*

**One Thousand Consecutive Deliveries under a Training for Childbirth Program.** THOMS, H., and WYATT, R. H. (1951). *Amer. J. Obstet. Gynec.*, **61**, 205. 8 refs.

This is a report on 1,000 deliveries of women who had been trained for childbirth according to methods advocated by Grantly Dick Read and now widely adopted in American clinics. Four years ago the nurses and physicians at the Thoms's Clinic in Newhaven were taught the exercise techniques which are now used in the clinic. An attempt is made to prepare women for childbirth psychologically and physically by means of a training programme in which they are taught the fundamentals of reproductive anatomy and physiology, and are trained also in relaxation techniques and muscle control to aid the natural forces of labour. In labour they are encouraged to use this technique by those in attendance, on whose sympathy and interest much of the success of the method depends.

Of the 1,000 women, 375 were primiparae and 625 multiparae. In 214 of the primiparae (73·3 per cent.) who underwent spontaneous delivery not over 125 mg. "demerol" (pethidine) or one dose of another agent was used. No analgesic was received in 19·9 per cent. In 530 (89·9 per cent.) of the spontaneous multiparous deliveries not over 125 mg. of pethidine or one dose of another agent was used and no analgesic was given in 49 per cent. No anaesthetic was given to 35·2 per cent. of the primiparae and to 46·3 per cent. of the multiparae. In primiparae the average total length of labour was 14·3 hours, in multiparae 8 hours.

Only eight infants (0·8 per cent.) were lost intranatally and neonatally (1,500 g. or over). The Authors believe that this low foetal loss is largely due to the minimal amounts of anaesthetic used and the relatively short duration of labour.

[In future reports it would be an advantage if comparable figures were given of the results obtained in the clinic before the Dick Read technique was introduced, for there are still centres, mainly in Britain, where doubt is expressed whether the methods advocated offer any advantages in regard to duration of labour, diminution of operative deliveries, and stillbirths and neonatal deaths. No such figures have yet been published.]

*F. J. Browne*

**Care, Feeding, and Fate of Premature and Full-Term Infants born of Tuberculous Mothers.** RATNER, B., ROSTLER, A. E., and SALGADO., P. S. (1951). *Amer. J. Dis. Child.*, **81**, 471. 22 refs.

From their study of 55 infants born of tuberculous mothers between 1943 and 1945 at the Sea View Hospital, New York, the authors confirm the work of Debré and LeLong in 1925, which showed that the causes of death of such infants are not hereditary or constitutional factors, but imperfect artificial feeding and poor standards of care. In a series of 260 births between 1933 and 1945 no case of congenital tuberculosis occurred. The authors give details of the methods of feeding both full-term and premature infants, the methods being founded upon the administration of evaporated milk, maltose, and dextrin, with added protein in the form of calcium caseinate where necessary, and vitamins C, A, and D.

It is emphasized that tuberculosis does not develop in these infants if they are removed from contact with their tuberculous parents; one infant did develop tuberculosis, but it was in contact with its mother; separation is essential. Tuberculosis in the mother tends to be associated with prematurity of the infant, due not to the tuberculosis itself but to the disturbed metabolism of the mother. No case of transplacental tuberculosis in the infants born of tuberculous mothers occurred in this series, although 17·6 per cent. of the mothers died of advanced tuberculosis shortly after delivery.

*John Sumner*

**The Median Episiotomy. Its Technic and a Review of 1,500 Consecutive Cases.** SMITH, S. H. (1951). *West. J. Surg. Obstet. Gynec.*, **59**, 102. 7 figs, 12 refs.

After reviewing the history of median episiotomy the author describes his technique for this operation. Seven illustrations show in detail the incision and its subsequent repair. It is necessary to perform the episiotomy before the head is crowned, thereby preventing damage to the underlying fascia and also reducing the risk of a third-degree laceration. The incision should be made in the exact midline of the perineum "severing the tissue with nibling snips" with the scissors. After the third stage is completed a Gelpe retractor is used to ensure good exposure. Number 00 or 000 chromic catgut is employed; the sutures in the vaginal mucosa must include the underlying fascia to prevent its retraction and therefore subsequent relaxation of the posterior vaginal wall. A subcuticular stitch for the skin of the perineum is used. In a third-degree laceration it is not necessary to suture the sphincter, but only the fascia surrounding the muscle. Early ambulation in all cases is prescribed.

The author reviews 1,500 cases of median episiotomy performed at the Mercy Hospital in San Diego; this number represents 54 per cent. of the total deliveries. Third-degree lacerations occurred in 28 instances (1·86 per cent.); all healed by first intention. The average stay in hospital was 4·9 days. *Jean R. C. Burton-Brown*

**Relationship of Armed Forces Research to Clinical Ophthalmology.** IMUS, H. A. (1951). *Sight-sav. Rev.*, **21**, 73.

**The Inheritance and Distribution of the Duffy Blood Groups.** RACE, R. R., HOLT, H. A., and THOMPSON, J. S. (1951). *Heredity*, **5**, 103. 1 fig., 7 refs.

**Report on a Country-wide Study of Silicosis in the Metal Mines of Japan.** YAMAMOTO, M. (1951). *Arch. Industr. Hyg. occup. Med.*, **3**, 339. 5 figs.

**Specific Dyslexia ("Congenital Word-Blindness"). A Clinical and Genetic Study.** [In English.] HALLGREN, B. (1950). *Acta psychiat., Kbh.* Suppl. 65, 1. 3 figs, bibl.

**Correlation of Epileptic Seizures, Electroencephalograms and Emotional State. Some Preliminary Observations in Several Children.** BERLIN, I. N., and YEAGER, C. L. (1951). *Amer. J. Dis. Child.*, **81**, 664. 3 figs, 7 refs.

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